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Inheritance of Resistance to Sheath Blight in Long Grain Rice (Genetics, Fungus Disease).

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INHERITANCE OF RESISTANCE TO SHEATH BLIGHT IN LONG GRAIN RICE

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INHERITANCE OF RESISTANCE TO SHEATH BLIGHT IN LONG GRAIN RICE

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ABSTRACT

The inheritance of resistance to the sheath blight disease of rice (*Oryza sativa* L.), caused by *Rhizoctonia solani* Kuhn, was studied using as parent lines four susceptible commercial varieties--Lebonnet, Labelle, Starbonnet and Leah, and three resistant lines: L201, RU7902185 and RU7902191.

The parental lines, F_2 and F_3 progeny of each cross were inoculated when the seedlings were 35 days old using the isolate LR172, and then kept in a humidity chamber for two weeks. The infection level of each plant was evaluated using a 0-9 rating system where 0 indicates no symptoms and 9 indicates the most severe infection. Infection height as a percentage of the total sheath height was also estimated.

The frequency distribution of the F_2 progeny of susceptible x resistant crosses showed a bimodal distribution with the modal classes at 5 and 7. The resistant parents had a modal class at 4 and the susceptible parents had their modal class at 7.

The distribution of the F_2 progeny of most of the crosses appears to fit a 9:7 resistant to susceptible ratio. This suggests that two pairs of complementary genes with a high level of dominance controlled resistance to sheath blight. The analysis of variance indicates a high level of dominance and epistasis. Rating disease reaction by measuring the height of the highest lesions was not a

satisfactory method for rating sheath blight infection since lesions sometimes occurred around the uppermost leaf collar on otherwise symptom-free plants.

Heritability estimates were low. The highest estimates were 16.09% and 24.62% using respectively the regression and correlation of F_2 on F_3 line means. Since sheath blight resistance appears to be an effect of complementary dominant genes, the epistatic interactions may be partly responsible for the low heritability. However the low heritability suggests a need for progeny testing of individual plants selected in segregating populations in order to effectively identify resistant genotypes.

INTRODUCTION

Rice (*Oryza sativa* L.) is the staple food of approximately half of mankind. Among the cereal crops, rice is second only to wheat in terms of world production. About 90% of the world's rice is grown in Asia (Chandler, 1979). African production represents only 2.5% of the total world output (Aw, 1978). In the Americas, the leading rice-producing country is the United States followed by Brazil. The major rice-producing areas in the United States are in Arkansas, California, Louisiana, Mississippi and Texas.

In the past sheath blight of rice, caused by *Rhizoctonia solani* Kuhn, has been reported to be a minor disease of rice (Ryker and Gooch, 1938). However during the last few years sheath blight has increased in prevalence and severity and has been recognized as a destructive disease of rice. It is now considered to be the second most important disease of rice in the United States, next to rice blast. It was estimated that the 1968 rice yield was reduced by 25% in Arkansas due to sheath blight (Templeton and Johnson, 1969). The disease became epidemic in the rice production areas of Southern Louisiana in 1971 and caused considerable loss to Louisiana rice growers (Rush, 1971).

Although some fungicides have given satisfactory control of sheath blight (Chien and Hung, 1971; Rush et al., 1976; Rush et al., 1977; Rush et al., 1982), chemical control may cause environmental

pollution and increase production costs. Therefore, the most economical and preferred method for controlling sheath blight remains the development of resistant varieties.

It should be noted that progress in plant breeding is dependent on the genetic information and the extent to which such information is utilized. Knowledge of the inheritance of resistance is very important to the breeder in order to develop an effective program for selecting resistant varieties. Information on the inheritance of resistance to sheath blight is limited. This study was therefore initiated to (1) evaluate procedures for selection of sheath blight resistant plants in early generation segregating populations; (2) investigate the inheritance of resistance to sheath blight; and (3) estimate the heritability of resistance to sheath blight.

REVIEW OF LITERATURE

Distribution and Importance of Sheath Blight

About forty diseases attack rice plants in the field. Of these the second most prevalent and important is probably sheath blight. It follows blast in causing yield losses. The fungus causing the disease has been known to be present in all parts of the world where its host plants are extensively grown (Houston, 1945).

Padwick (1950) reported that damage to rice plants due to sheath blight has increased since it was first described by Miyake in 1910. The disease has been recorded since then in various Asian countries including Taiwan, China, India and Ceylon. Sheath blight was thought to occur only in Asia, but recently it has been reported in Surinam, Venezuela, Madagascar (Ou, 1972) and Brazil (Amaral and de Jesus, 1973). The wide distribution of sheath blight in China has been reported by Ling (1948). In the rice regions of the southern parts of the Yangtze Valley in China, its incidence has recently increased with the growing of broadleaved semi-dwarf varieties and the adoption of close planting (Sun, 1980).

In the United States, sheath blight has been found in Louisiana, Texas, Arkansas, California and Florida (Tullis, 1934; Ryker and Gooch, 1938; Atkins, 1974). The pathogen has been endemic for many years, but recently the disease has become a serious problem to rice, especially in fields of long grain rice (Marchetti, 1983).

The yield loss from the disease depends on the age of the plant at infection and the extent to which the disease develops. Loss in yield was significant in early rice varieties (Padwick, 1950; Singh and Pavgi, 1969). According to IRRI (1975), a yield loss of 25% at the lower nitrogen level and 20% at the higher nitrogen level was recorded when all the leaf sheaths and leaves are infected. Tsai (1974) studied the yield losses in early maturing variety IR 833-6-2-1-1 due to sheath blight at different stages of inoculation. The reduction in yield was 7.95, 7.15, 10.78 and 11.73% at 15, 30, 60 days after seeding and at booting stage respectively.

Sheath blight caused by *R. solani* Kuhn may be responsible for considerable loss in milling quality of the affected rice, especially when infection occurs early and plants are killed before grains are completely filled (Templeton and Johnson, 1969). Mizuta (1956) cited by Ou (1972) estimated that 20% reduction in yield loss may be incurred if the disease develops up to the flag leaf. Rush (1971) obtained a difference of 1266 lb/a between diseased and healthy plants. He also reported a yield loss of 33% due to sheath blight. Similar results were obtained by Lee and Courtney (1982a) who reported 17.5% and 33.5% yield reduction due to sheath blight respectively in Starbonnet and Lebonnet varieties.

In Japan sheath blight infected about 120,000 to 190,000 ha, resulting in a loss of 24,000 to 38,000 tons of rice annually (Ou, 1972; Tsai, 1974). Recently Hashiba (1984) reported that in Japan the area affected by sheath blight disease averaged from 1.2 to 1.4

million ha, or about 32 to 50% of the total rice cropping area. In the United States the most commonly grown rice varieties are susceptible to this disease with yield losses occurring in the general magnitude of 25-50% in Lebonnet, 15-25% in Starbonnet and 7-15% in Mars (Lee and Courtney, 1981). Little yield losses occurred when the disease developed even severely, late in the season after the grain filling was nearly complete (Hunter et al., 1977).

Taxonomy and Description of the Fungus

The correct nomenclature of the sheath blight fungus has long been in controversy because of the variability of the species. English and American pathologists have used the name *Corticum vagum* Berkeley and Curtis, whereas European workers refer to the same fungus as *Corticum solani* Prilleux and Delacrois (Houston, 1945).

The sheath blight pathogen has been described and designated as *Trichoderma lignorum* (Tode) Hartz by Tullis (1934), *Sclerotium irregulare* by Miyake (Padwick, 1950), *Corticum sasakki* (Ou, 1972), *Hypochnus sasakii* Shiroi, *Pellicularia filamentisa* by Rogers (1943), and *Rhizoctonia solani* by Kuhn (Manibhushanrao et al., 1979). However, Roger's nomenclature was rejected by Venkatarayan (1949) and Talbot (1965). The name *R. solani* Kuhn, the imperfect state, is the most commonly used.

Exner (1953) reported that among the genus of *Rhizoctonia* found in Louisiana, four species are responsible for disease of considerable importance:

- *Rhizoctonia microsclerotia* Matz which causes web-blight of beans and other plants;
- *Corticum sasakii* Matsumoto, causative agent of sclerotial disease of sugarcane, rice and certain grasses;
- undescribed *Corticum* which is responsible for leaf blight of figs;
- *R. solani* which is responsible for root and stem disease of many plants.

The perfect stage of *R. solani* Kuhn is presently considered to be *Tanatephorus cucumeris* (Frank) Donk. The basidial stage of the sheath blight fungus (*Tanatephorus cucumeris*) rarely has been reported to occur on rice in the field (IRRI, 1973). Parmeter and Whitney (1970) characterized *R. solani* by:

- pale to dark brown, rapidly growing mycelium of relatively large diameter;
- production of sclerotia of nearly uniform texture and varying size and shape, often less than one millimeter diameter;
- pathogenecity to a wide range of hosts;
- possession of a prominent septal pore apparatus and multinucleate cells in actively growing hyphae.

Studies of the vegetative cells incidate that they are multinucleate. According to Sanford and Skoropad (1955), the number of nuclei per cell varies from 4-25 with 4-8 in the tip cells and 6-11 in older vegetative hyphae. Nuclei measure 3.1 by 2.0 μ and are oval, but change shape as they move in the cells (Flentje et al., 1961). Saksena (1962) observed the division of occasional living

nuclei, implying that individual nuclei divide independently. He concluded that nuclear division in the vegetative cells was unlike the classic mitotic division of nuclei in higher plants.

The fungus grows readily on various common media. When the mycelium is 6-8 days old, it starts to produce sclerotia which are round in shape, 1-5 mm diameter and composed of compact masses of hyphal cells, brown to dark brown in color. With increasing age the hyphae become brown with infrequent septations (Ou, 1972).

Hashiba et al. (1972) reported that sclerotia at an early stage of development sank in water; later they became buoyant. They also stated that buoyancy was closely associated with the number of empty cells in the outer layer. According to Hashiba and Mogi (1975) cells of the outer sclerotial layer that became empty with age were formed as a result of the differentiation of the sclerotial cells during sclerotial formation. Sclerotia that float on water play very important roles in the outbreak of the sheath blight of rice plants.

Butler and Bracker (1970) stated that in addition to ordinary vegetative hyphae, *R. solani* produced simple or branched chains of short, broad, barrel-shaped, irregular brown cells.

Differences in pathogenicity of isolates have been recorded by Akai et al. (1960). The isolates with no pathogenicity showed a poor mycelial growth while pathogenic ones showed rapid growth of mycelium as much as 8-11 mm per 5 hours. Hashiba et al. (1984), working with three isolates of anastomosis group 4 (AG-4), reported

that weakly pathogenic isolates of *R. solani*, showing slow growth, contained the plasmids, but pathogenic isolates showing normal growth, contained no detectable plasmid DNA. Chien and Jong (1963) classified 300 isolates into seven culture types and six physiological races based on the disease actions of 16 varieties. However, the susceptible and resistant reactions used in separating the races were not so clear-cut.

Manibhushanrao et al. (1979) described the basidial state as a white powdery or frosty layer on healthy leaf sheaths and occasionally on leaves. The basidia are barrel-shaped or clavate, bearing 2 to 4 sterigmata which arise as blunt knobs and later become horn-shaped. Hawn and Vanterpool (1953) reported that basidia were simple clavate with an average measurement of 13.4 x 8.1 μ, and the basidiospores thin-walled, smooth, hyaline and apiculate with an average measurement of 8.9 x 5.5 μ. The size of 12.4 x 8.3 μ for basidia was recorded by Exner (1953); 10-15 x 7-9 μ for basidia and 8-11 x 5-6.5 μ for basidiospores by Ou (1972); 10-15 x 7-9 μm for basidia and 8-11 x 5-6.5 μm for basiodiospores by Manibhushanrao et al. (1979). The means of 100 randomly selected single basiospores collected from sorghum measured 7.02 x 8.71 μ (O'Neill, 1976). Exner and Chilton (1943), studying the cultural differences among single basiodiospore isolates, concluded that some type of segregation occurs in the formation of basidiospores and that different strains of *R. solani* arose as a result.

Physiology of the Fungus

The occurrence and severity of a plant disease depends upon the interaction of many factors in the physical and biological environment. Several investigators have reported different temperatures for optimum growth of the fungus. According to Richards (1921), much damage was produced between 15°C and 21°C while 18°C was most favorable for tissue destruction and growing-point injury. Palo (1926) reported that sclerotial cells germinated within 12 hours at laboratory temperature ranging from 26°C to 30°C. At that temperature germ tubes protruded through the septum of the cells. Hemmi and Endo (1934), studying the effect of temperature on the infection of rice plants by the sheath blight fungus, concluded that the minimal periods of continuous wetting necessary for the infection of rice under experimental conditions were about 18 hours at 32°C and 24 hours at 28°C. At 36°C, infection seemed to be barely possible. The optimum temperature for the fungus was reported by Wei (1934) to be somewhere near 27°C, with the minimum about 8°C and maximum slightly higher than 37°C.

According to Nisikado and Hirata (1937), agar-slant cultures remained viable at least 13 months when stored at 0°, 5°, 10°, 15° and 25°C but were dead by 4-6 months at 30°C and 1 month at 35°C. When cultured on rice straw, the longevity was 11 months at 30°C and 4 months at 35°C. Wu (1965) found that the optimum temperature for growth of *R. solani* ranged between 25°C and 31°C. He also reported that the growth, pathogenicity and toxin production of *R. solani* were

found to be directly correlated when the cultures were incubated at different temperatures. The optimum temperature for mycelial growth was reported by Ou (1972) to be 30°C, maximum 40-42°C, with little or no growth occurring at 10°C. However, Ogoshi (1972) found that most isolates belonging to the group AG-1 grew rapidly at 28°C, but slightly at 35°C, and most isolates of the group AG-2 grew rapidly at 23-25°C but not at 33°C.

Sheath blight disease is known to be especially destructive under highly humid and warm temperature. For sclerotia germination, high relative humidity above 95-96% was required (Ou, 1972). Habisha et al. (1982) reported that the vertical development of sheath blight lesions was the highest at 100% relative humidity and lowest at 86% relative humidity.

The effect of light on the fungus has been studied by several researchers. Kotila (1929) stated that light stimulated sporulation in strain R40 of *Tanatephorus cucumeris*, but some sporulation occurred in the dark. According to Hemmi and Endo (1932) sclerotia were formed more abundantly in the presence than in the absence of light, and that their formation was also accelerated by a sudden fall in temperature. However, continuous fluorescent light was found to suppress vegetative growth of *R. solani* (Butler, 1957). This was supported by Whitney (1964) who stated that the strain R43 grew and sporulated vigorously in the dark. Both authors suggested that insufficient aeration accounted for the partial or complete absence of sporulation.

A slightly acid reaction appeared to be most favorable for sclerotial formation (Sherwood, 1970). Most rapid growth usually occurred on media at pH 5.0-7.0.

Emphasis has been placed on the nutritional status of the inoculum when evaluating the pathogenic capabilities of *R. solani*. In infected host tissue, *R. solani* was capable of attacking hypocotyls in field soil. However, after a relatively short period in soil, the fungus, although viable, was unable to parasitize the plant without a supply of external nutrients (Kamal et al., 1967). Tu (1968) reported that the type, quantity of sclerotia and discoloration of the media were among the most variable characteristics affected by a nutrient deficiency. Weinhold et al. (1969) found a direct relationship between the concentration of carbon or nitrogen source in the media and the virulence as determined by the area macerated tissue. Hashioka and Yamada (1981) concluded that sclerotia of *R. solani* formed on rice plants were more resistant to high temperature and high humidity levels than those formed on potato-sucrose-agar (PSA). These sclerotia had high proportions of "empty" cells in their outer layer.

Aoki et al. (1963) suggested that phenylacetic acid was produced at first by *R. solani* and then metabolized to its hydroxy derivatives, and these acids played together a main part in the pathogenic actions of the fungus. Glucose appeared to interfere with disease development through the prevention of symptoms (Weinhold and Bowman, 1974). However, the glucose did not interfere with

host-pathogen specificity expressed by infection-cushion formation. Moromizato et al. (1980) reported that inhibitive amino acids could namely reduce the lateral branching of hyphal interweaving which is necessary for sclerotial development. They also reported that inhibition of the sclerotial cell enlargement during the maturation of sclerotia resulted in the limitation of the final sclerotium size and weight.

Disease Cycle

Sclerotia are the main source of inoculum. Critical observations made by Singh and Pavgi (1969) on recurrence and development of the disease over several years indicate that the loosely attached sclerotia from infected plants fall on the soil during harvest and act as the source of primary inoculum for the succeeding crop. The sclerotia of the fungus can survive in the soil from several months to one or more years, depending on the temperature and moisture content. While the soil is being prepared for rice planting, the sclerotia float on water and infect plants with which they come in contact (Ou, 1973). In natural soil, *R. solani* grows less vertically than horizontally. A speed of growth of more than 1 cm per day was recorded by Blair (1943).

R. solani is carried on and in true seeds (Baker, 1947). Such transmission is significant because: (a) it introduces strains of the fungus to new areas or fields; (b) it contaminates disinfected soil, causing severe seedling loss; (c) it assures the continued

association of a virulent strain of the fungus with the appropriate host. The fungus may be carried away as mycelia or sclerotia, and come in contact with rice plants and start infection (Ou, 1972).

The fungus overwinters as sclerotia or mycelia in soil in association with plant residues (Endo, 1932; Mgonja et al., 1982). The sclerotia are easily detachable from the host. They fall, sink and soon float to the water surface as they fully mature (Manhibushnrao, 1979). Kozaka (1965) reported that the mycelium entered the plant through the stomata or it penetrated directly through the cuticles. Usually the fungus enters the leaf from inner surface, but may enter through the surface of the blade. After the primary lesions are formed, mycelia grow rapidly on the surface of the plant and inside the tissues to initiate secondary lesions (Ou, 1972).

Control of Sheath Blight Disease

Successful control measures are determined by the characteristics of the pathogen, the host crops and the environment. In rice fields various wild grasses growing in mixture with rice may serve as a source of infection (Atkins, 1958). Sheath blight pathogen can also attack a large number of plants. Kozaka (1965) reported that the sheath blight pathogen infected 188 species in 32 families. Eighteen species of *Oryza* were reported to be affected by sheath blight.

The ideal control measure would be to exclude the pathogen from areas or fields where it does not now exist. Palo (1926) suggested that in badly infected fields dried plant materials should be burned in order to destroy the sclerotial bodies. After a heavy rain the soil should be plowed, harrowed and pressed with a roller. The burried sclerotial bodies will be killed within five or six months. According to Lee and Courtney (1982b), rice straw burning reduced the number of sclerotia from 193 to 97 per square feet. They also indicated that the three year average sclerotia viability of 60 percent was reduced to 30 percent.

Crop rotation, one of the methods used to control disease, was reported by Palo (1926) and Wei (1934) to be ineffective in controlling *R. solani* since the fungus is able to infect many other crops beside rice. However, Leach and Gardner (1970) mentioned crop rotation as one method for controlling disease. The beneficial results of these cultural practices may be due to the effects on pathogen nutrition rather than to a reduction in pathogen population (Weinhold et al., 1969). Results obtained by Chu (1966) indicated that *R. solani* was far more abundant in the cultivated soil than in fallow. In the opinion of some investigators (Palo, 1926; Wei, 1934; Rush, 1972) control can be improved by avoiding close planting and heavy grassy weed infestations. Since fungi causing the disease overwinter in the soil and plant debris, rice should not be grown in successive seasons in fields where severe infestations have occurred in the previous crop (Rush, 1972).

Commercial varieties are susceptible to sheath blight. This led researchers to look for effective chemical control of the disease. Several fungicides have shown promising results. In greenhouse tests, Chien and Hung (1971) obtained good results with benlate. In the field, significant yield increases were reported with benlate 50WP, Duter (Rush et al., 1976; Rush et al., 1977; Rush et al., 1980; Lee and Courtney, 1981), with NTN 19701 (Rush et al., 1982, Whitney, 1982) and with CGA 64250 (Rush et al., 1981; Lee and Courtney, 1982a). Good results in controlling sheath blight were obtained with combinations of fungicides (Rush et al., 1980; Rush et al., 1982).

Recently it has been reported that the anti-fungal antibiotic, Polyoxin, produced by *Streptomyces cacaoi* var. *asoensis* was as effective as the organoarsenial compounds (Sasaki et al., 1968). According to Henis et al. (1978), PCNB at 4 µg/g soil added with Trichoderma inoculum had an additive effect on the disease control and a synergistic effect on the decrease in inoculum density of *R. solani* propagules. In the opinion of Elad and Hadar (1981) *Trichoderma harzianum* preparation reduced the incidence of stem rot on carnations caused by *R. solani* by 70% when applied at the rate of 150 grams (dry weight) per square meter.

Varietal Resistance to Sheath Blight

Various screening techniques have been used to determine differences in varietal resistance to sheath blight. Hashioka

(1951), screening about 200 hundred varieties under greenhouse and field conditions, concluded that the difference in resistance among the variety-groups was more distinct when inoculation was made with the plants at the booting-heading stage than with seedlings. Chien and Jong (1963) used different varieties of rice to identify the reactions of six physiologic races of sheath blight pathogen. They reported that the *indica*-type varieties were more resistant than the *japonica*-type varieties. Tests conducted at IRRI (1966, 1972, 1980), showed that very few varieties were resistant to sheath blight. From 470 varieties tested, only 29% were moderately resistant, 65% were intermediate and the remaining were susceptible. The number of sclerotia counted on 1979 varieties was positively correlated with the degree of sheath infection ($r = 0.8$).

Resistance to *R. solani* is often associated with maturation of host tissues. Before the heading stage, the upper leaf sheaths and blades are more resistant than the lower ones, but after heading the susceptibility of the upper parts increases with increasing plant age (Ou, 1972). Results obtained by Toriyama (1972), from inoculation tests in the seedbox, indicated that late maturing varieties had more resistance than early ones. Kush (1977) reported distinct varietal differences with various methods of inoculation but the results did not completly agree. From a sample of over 1,000 varieties and lines screened at IRRI, several moderately resistant lines were identified. Variety Ta-poo-cho-z showed resistance at

all stages while variety DA29 was resistant and moderately resistant, respectively, at seedling and late stages.

There are few reports to suggest that there is a wide variation in the resistance of rice varieties to sheath blight but it is difficult to find a completely resistant variety. In a screening trial with six exotic high-yielding varieties and 14 local improved varieties of rice, Das (1970) found only NC678 and Dudsar resistant and the remaining were moderately resistant and moderately susceptible. Roy (1977), after screening 135 varieties, did not find any resistant ones. Field response of rice varieties and lines to infection by *R. solani* have been reported by several investigators. At the Rice Experiment Station, Crowley, from 600 lines and varieties screened per year, only 17% were resistant in 1975 (Hoff et al., 1975), 168 lines were resistant in 1976 (Hoff et al., 1976) and 122 lines showed resistance in 1977 (Rush et al., 1977). The 1979 disease nursery planted at the Crowley Experiment Station indicated that 74 entries of 560 tested showed resistance to sheath blight (Hoff et al., 1979). Tests conducted in the greenhouse using 200 entries indicated that Zenith, Mars, Taducan and Tetep were among the most resistant (Rush et al., 1980). In a special sheath blight nursery, 63 lines were resistant to sheath blight. They included six long grain type lines which were considered to have multiple disease resistance (Rush et al., 1981).

In 1982, Rush et al. (1982) obtained 32 long grain type lines resistant to sheath blight out of 681 entries tested. Among

them, only six lines showed multiple resistance to four diseases including sheath blight. Teng and Hsu (1979) reported that the relatively resistant varieties tended to have a taller plant height, and semidwarf rice varieties tended to be more susceptible to the disease. They also reported that the reactions of rice varieties to sheath blight and brown plant hoppers were positively correlated.

Histological aspects of infection by *R. solani* on rice cultivars differing in resistance levels have been studied by Marshall and Rush (1980a) and on cotton by El-Samra et al. (1981). A highly significant correlation was found between disease severity ratings of the cultivars and both infection cushion formation ($r = 0.970$) and lobate appressoria (Marshall and Rush, 1980a). The nature of infection cushion formation by *R. solani* on rice cultivars with different levels of resistance to sheath blight has also been studied by Marshall and Rush (1980b). They concluded that cultivars resistant to the disease had abundant wax deposits and intermediate cultivars produced varying amounts of wax deposits. No wax deposits were produced by susceptible cultivars. Stockwell and Hanchey (1984), studying the role of the cuticle on resistance to *R. solani*, suggested that cuticle thickness plays a more important role than calcification of cell walls in the resistance of older plants.

The determination of mode of inheritance of resistance to sheath blight appeared to be difficult as a wide range of variations in disease symptoms affected the classifications of plant reactions. Hashioka (1951) reported that resistance to sheath blight was

inherited as a dominant character, and in crosses between resistant and susceptible varieties the majority of the F_2 populations were resistant. Workers at the Taiwan Agricultural Research Institute obtained a 3:1 ratio in F_2 populations in some crosses (Chang, 1962) while Hashioka (1951) found a bimodal distribution of F_2 progenies with resistance dominant over susceptibility. Masajo (1976) concluded from his study that resistance to sheath blight was partially dominant over susceptibility. He also reported that resistance to sheath blight was controlled by as few as two pairs of genes. Inheritance of the ability of a cultivar to support infection cushion was studied by Marshall (1979). The results indicated that the character was inherited by one or two pairs of genes with resistance dominant over susceptibility.

MATERIALS AND METHODS

The experimental materials consisted of four susceptible varieties--Lebonnet (LBNT), Labelle (LBLE), Starbonnet (STBN) and Leah, and three resistant or moderately resistant lines or varieties: L201, RU7902185, RU7902191. These varieties and lines were selected based on the sheath blight disease rating in the 1979 disease nursery at Crowley, Louisiana (Table 1).

All the seeds were supplied by the Department of Agronomy, Louisiana State University. In the fall of 1982, the crosses were made in the greenhouse. To match the different dates of flowering of resistant and susceptible varieties and lines, seeds were planted in pots at 10-day intervals.

At flowering, panicles to be crossed were selected. Then all florets from the top of the selected panicles that had undergone anthesis and all young florets from the bottom of the panicles were removed. With scissors, one-third to one-half of the florets were cut away obliquely to expose the anthers. Using a small vacuum emasculator, the six anthers were removed. The emasculated panicles were then covered with transparent paper bags to prevent pollination by a foreign rice plant. The next morning, as anthesis began, panicles of the male parents were cut and carried to the emasculated panicles. After the pollen was dusted over them, the female panicles were covered again with transparent bags. The pollinated

Table 1. Identification, agronomic characteristics and disease reaction of the plant materials selected for the inheritance study.

Parental lines	Variety or line	CI or RU number	No. of days to heading	Disease Reactions[1]			
				Rhizoct. solani	*Helm. oryzae*	*Cercosp. oryzae*	*Entyloma oryzae*
P_1	Lebonnet	CI9882	78	8	4	5	5
P_2	Labelle	CI9708	73	8	4	5	8
P_3	Starbonnet	CI9584	98	7	3	6	2
P_4	Leah	--	78	6	2	7	4
P_5	L201	CI9971	72	2	5	0	5
P_6	--	RU7902185[2]	91	2	3	0	4
P_7	--	RU7902191[2]	97	2	3	0	0

[1]Based on a 0-9 scale, where 0 indicates no infection of disease and 9 indicates the most severe infection.

[2]RU7902185 and RU7902191 are two sister lines having the following pedigree: S242/UNKN/3/RXRE/R252//TN-1/4/RRTL/3/6001/CENT//RXDL/SADR.

panicles were tagged and labelled. Twenty-five to 30 days after pollination, the F_1 seeds were ready for harvest. The crosses which succeeded are shown in Table 2.

After the F_1 seeds were harvested, they were treated in the oven at 50°C ± 2°C for four days to break the dormancy. Seeds of each cross were surface sterilized, germinated in petri dishes and then transferred into pots. For surface sterilization, seeds were placed in glass vials and a mixture of 2 parts 95% ethanol, 4 parts 5.85% sodium hypochlorite (bleach) and 10 parts distilled water was added to cover the seeds. The vials were agitated for 5 minutes, followed by a rinse for 10 minutes in distilled water.

The pots contained a soil mixture consisting of 1 part steam sterilized Oliver silt loam soil, 1 part sand and 1 part peat moss by volume. Each pot contained approximately 3 lbs of soil mixture. At maturity, the F_2 seeds were harvested and stored.

Evaluation of F_2 Populations to Sheath Blight

The F_2 seeds from each cross and parent were planted in galvanized flats containing approximately 10 kg of steam-sterilized Oliver loam soil. The flats were 55 cm long, 40 cm wide and 10 cm deep and were arranged on two benches covered with polyethylene to make them water tight. Each bench measured about 6.47 m long, 1.24 m wide and 0.18 m deep and held 30 flats. The seeds were planted at the rate of 10 seeds per row of flat and 6 rows per flat,

Table 2. List of successful crosses used in the study.

Cross	Type of Cross
LBNT x LBLE (P_1 x P_2)	Susceptible x Susceptible
LBNT x RU7902185 (P_1 x P_6)	Susceptible x Resistant
LBNT x RU7902191 (P_1 x P_7)	Susceptible x Resistant
LBLE x L201 (P_2 x P_5)	Susceptible x Resistant
L201 x LBLE (P_5 x P_2)	Resistant x Susceptible
LBLE x RU7902185 (P_2 x P_6)	Susceptible x Resistant
LBLE x RU7902191 (P_2 x P_7)	Susceptible x Resistant
STBN x RU7902185 (P_3 x P_6)	Susceptible x Resistant
STBN x RU7902191 (P_3 x P_7)	Susceptible x Resistant
LEAH x RU7902185 (P_4 x P_6)	Susceptible x Resistant
LEAH x RU7902191 (P_4 x P_7)	Susceptible x Resistant

making a total of 60 plants per flat. The rows were 55 cm long and were spaced approximately 6 cm apart.

All seven parental lines and F_2 progeny of 11 crosses were planted. Each parent had 20 plants and each cross 60 plants per replication. The treatments were divided into entries of 20 plants, such that each parent constituted one entry and each cross three entries. A total of 39 entries were randomized in each replication. The entire experiment was conducted in a randomized block design with four replications arranged on two benches. A total of 2,699 plants, including 469 parental plants and 2,230 F_2 plants, were individually evaluated.

After planting, the benches were water-filled to a depth of approximately 8 cm. To insure the same number of plants for each entry, seeds of the same material were planted in extra flats. One week after germination, these seedlings were transplanted in the experimental flats, replacing the non-germinated seeds.

Fertilizers were applied at the rate of 25 kg N, 25 kg P_2O_5 and 25 kg K_2O per ha when the seedlings were 35 days old. This was done by preparing a mixed fertilizer solution containing N, P, K and applying 165 ml of the solution diluted in 10 liters of water per bench. A sprinkler can was used to get a uniform application.

Shortly after the seeds were planted the preparation of *R. solani* inoculum was begun in the laboratory of the Department of Plant Pathology of Louisiana State University. The fungi were grown on a potato dextrose agar (PDA) medium which was prepared by adding

500 ml of distilled water to 19.5 g of PDA powder in a 1,000 ml flask. The flask was shaken and autoclaved for 15 minutes at 121°C and 15 psi. The medium was poured into petri dishes. After cooling, a piece from a pure culture of *R. solani* isolate LR172 was cut and transferred to each petri dish. These petri dishes were then held at room temperature (24°C) for 7-10 days for multiplication of the fungi.

To prepare the inoculum rice grain and rice hulls were mixed in a ratio of 1 part rice grain to 2 part rice hulls by volume and 1,600 ml of this mixture was placed in 2,000 ml flasks. Water was added until it reached the 800 ml level marked on the flasks. The flasks were capped with cotton and wrapped with aluminum foil to minimize contamination, and then autoclaved for 15 minutes at 121°C and 15 psi. They were left for 24 hours to cool and autoclaved for the second time. They were then placed on a laminar air flow hood. The surface was sprayed with alcohol and cleaned with paper. An ultraviolet light was turned on for five minutes or more to kill any bacteria. After these operations, the mouth of the flasks and the inoculating needles were sterilized in a flame. Using the sterilized inoculating needle, a plug of mycelium and agar was taken from the pure culture of the fungus in the petri dishes and transferred to the flasks. The flasks were plugged again with cotton and wrapped with foil. The flasks were then incubated at room temperature and approximately two weeks after the flasks were inoculated, the medium was completely

permeated by the fungi. Forty days after the rice seeds were planted the rice seedlings were inoculated with the rice grain-rice hull-fungi inoculum previously prepared.

Prior to inoculating the seedlings, a section of PVC pipe which had been cut in half was placed between the rows of seedlings. The white PVC pipes were selected for this use after a toxicity test. In the test the white pipe showed no toxic effect on the *R. solani* fungi. The pipe sections were 4 cm in diameter and 47 cm in length. The pipes were washed and placed between the rows of rice seedlings in the flats. The use of the PVC pipes allowed more contact between the plant and a given amount of inoculum, thereby decreasing the quantity of inoculum needed per plant. This procedure also minimized the soil contact of the inoculum, decreasing the moisture uptake of the inoculum through contact with the soil.

The seedlings were inoculated by placing 5 ml of grain-hull inoculum at the base of each seedling. After inoculation, a frame over the benches was covered with clear polyethylene plastic to create a humidity chamber over the inoculated seedlings. The humidity chamber was 6.47 m long, 1.24 m wide and 0.95 m high. Humidity in the chamber was increased in winter by using a sprinkler once in 24 hours for 6 minutes. Two weeks after inoculation, the polyethylene covering was removed and the seedlings were allowed to dry for three to five days before being rated for sheath blight infection.

The reaction of each plant to sheath blight was rated using the following scale:

0 Plants healthy, so symptoms.

1 Lesion centers gray-green to nearly white, margin of lesion a broad red-brown or purple-brown border usually broader than necrotic center, less than 2.5% of the tissues affected.

2 Few oval or coalesced lesions on lower sheaths or at infection points, lesions with broad red-brown border 5% or less of tissues affected.

3 Lesions on lowest sheath with narrow, red-brown border, coalescing, less than 10% of tissues affected.

4 Lesions mainly restricted to sheaths on lower third of plant, lowest leaves, lesions discrete or coalescing with narrow red-brown border, 10-15% of leaf and sheath tissues affected.

5 Lesions mainly restricted to sheath and leaves of lower half of plants, lesions usually coalescing with large necrotic centers and narrow red-brown borders, 15-25% of tissues affected, culm not injured.

6 Lesions usually coalescing and affecting lower two-thirds of the sheath area of plant, lesions extending to blades of lower leaves or lower leaves killed by injury to sheath, 25-40% of tissues affected.

7 Lesions usually coalescing and affecting lower three-fourths of sheath area of plant, lesions extending to leaf blades of lower two-thirds of the plant, 40-60% of tissues affected, outer portion of culm may be brown.

8 Lesions reaching the uppermost leaf, lower sheaths with coalesced lesions covering most of tissue, lower and middle leaves dead or dying, 60-80% of tissues affected.

9 Lesions reaching the uppermost leaf, lower leaves mostly dead, sheaths dried, culms brown.

In addition to rating the seedlings for sheath blight infection on a 0-9 scale, the height of infection as a percentage of the total sheath height of each seedling was computed using the following formula.

$$\text{Height of infection} = \frac{\text{height of uppermost sheath blight lesion}}{\text{total sheath height}} \times 100$$

The height of the uppermost lesion on a seedling was measured as the level of sheath blight infection even if the infection was spotty and sometimes limited to a few lesions on the upper portion of the sheath. The total leaf sheath height was measured from the base of the seedling to the height of the uppermost leaf collar.

A second test with F_2 seedlings was conducted using the procedure described above with the exception that the polyethylene humidity chamber was removed when seedlings of the most susceptible varieties were killed, which was four weeks after inoculation. The rating of each seedling was made using the 0-9 scale previously described.

Evaluation of F_3 Populations

The F_3 generation of the following four crosses were evaluated for their resistance to sheath blight:

1. LBNT x RU7902185
2. STBN x RU7902191
3. LBLE x L201
4. LBNT x LBLE

One hundred F_2 plants were selected at random from each cross to produce F_3 progeny.

The F_3 seeds were harvested and treated at 50°C ± 2°C for four days to break the dormancy. The F_3 lines, and F_1 and parental lines' seeds were planted in flats. One week after germination the seedlings were thinned to 20 plants per row. The experiment was conducted as a randomized block design with two replications. Each flat contained 6 rows spaced about 5 cm apart. The plants were fertilized at the rate of 25 kg N, 25 kg P_2O_5, 25 kg K_2O per ha. A day before inoculation, the white PVC pipes were placed between the rows of plants. Thirty days after planting the seedlings were inoculated with the rice grain-hull inoculum prepared following the same procedure as in the F_2 test. A portion of the inoculum (5 ml) was placed at the base of each seedling. Immediately after inoculation, the polyethylene humidity chamber, the same size as the one described previously, was installed over each bench. Fifteen days after inoculation, the poylethylene sheet was removed and the seedlings were allowed to dry for three to five days. The reaction of each plant to sheath blight disease was taken using the 0-9 scale previously described. The polyethylene humidity chamber was then closed to allow the fungus to grow again. The humidity chamber was removed two weeks later when most susceptible parents were almost killed or had a score of 9. After three to five days, a second reading was taken, also following the 0-9 scale.

Statistical Procedure

Only data from the 0-9 rating method were used for the statistical analysis and for estimating heritability.

The model. The experiment was intended as a diallel cross. The particular crosses for which data are available represent the combined effects of limited material and poor seed set. They do not conform to any standard design. If $P_1 \times P_2$, $P_2 \times P_5$ and $P_5 \times P_2$ are omitted, then the remaining crosses are all possible crosses of two resistant lines (P_6 and P_7) used as male parents to four susceptible lines (P_1, P_2, P_3 and P_4) used as female parents. Line P_5 is also resistant, but is not represented in crosses with any line other than P_2. Since general combining ability of P_5 is completely confounded with the specific combining ability of $P_5 \times P_2$, the two crosses involving P_5 only give information about the possible presence of reciprocal effects. The most reasonable analysis under the circumstances is to analyze the susceptible x resistant crosses (excluding P_5) as a North Carolina 2 (NC2) design (Comstock and Robinson, 1948) and discuss the three remaining crosses separately.

The model for analysis can be written according to Becker (1975) and Hallaeuer and Miranda (1982) as follows:

$$Y_{ijk} = \mu + M_i + F_j + (MF)_{ij} + R_k + e_{ijk} \qquad (1)$$

where Y_{ijk} is the observation of the full-sib progeny mean in a plot in the k^{th} replication of the j^{th} maternal plant and the i^{th} paternal plant.

μ is the overall mean.

M_i is the effect of the paternal plant.

F_j is the effect of the j^{th} maternal plant.

$(MF)_{ij}$ is the interaction of the paternal and the maternal plants.

R_k is the effect of the k^{th} replication.

e_{ijk} is the random error.

M_i, F_j and e_{ijk} are random with mean 0 and variances equal, respectively to σ_m^2, σ_f^2 and σ_e^2. P_1, P_2, P_3, P_4 and P_6, P_7 are random samples from the susceptible and resistant population of long grain rice varieties and lines.

Estimation of the components of genetic variance. The sums of squares are determined by the following formulas (Becker, 1975).

$$CF = Y_{...}^2/rmf \qquad (2)$$

where CF is the correction factor.

$Y_{...}$ is the total sum of all mean observations.

r is the number of replications.

f is the number of maternal lines.

m is the number of paternal lines.

$$SS_m = \sum_{i=1} Y_{i..}^2/rf - CF \qquad (3)$$

where SS_m is the sum of squares of the parental lines.

$Y_{i..}$ is the sum of the mean observation of the i^{th} parental line.

$$SS_f = \sum_{j=1} Y^2_{.j.}/rm - CF \qquad (4)$$

where SS_f is the sum of squares of maternal lines.

$Y_{.j.}$ is the sum of mean observation of the j^{th} maternal line.

$$SS_{mf} = \sum_j \sum_j Y^2_{ij.}/r - \sum_j Y^2_{.j.}/rm - \sum_i Y^2_{i..}/rf + CF \qquad (5)$$

where SS_{mf} is the sum of squares of the male x female interaction.

$Y_{ij.}$ is the sum of the mean observation of the i^{th} paternal and j^{th} maternal lines.

$$SS_r = \sum_k Y^2_{..k}/mf - CF \qquad (6)$$

where SS_r is the sum of squares of the replications.

$Y_{..k}$ is the sum of plot means of the k^{th} replication.

$$SS_e = \sum_k \sum_i \sum_j Y^2_{ijk} - \sum_i \sum_j Y^2_{ij}/r - \sum_k Y^2_{..k}/rm + CF \qquad (7)$$

where Y_{ijk} is the mean observation of the i^{th} paternal line, j^{th} maternal line, in the k^{th} replication.

The analysis of variance is shown in Table 3.

Assuming that the male effect and the female effect are random, we can write the following expected mean squares (Table 4). The components of genetic variance can be estimated using covarinaces of relatives (Hallauer and Miranda, 1982). From Table 4, these estimates can be computed as follows:

Table 3. Analysis of variance used in NC2 design.

Sources	df	SS	MS
Replications	$r - 1$	SS_R	$SS_R/(r - 1)$
Males (resistant)	$m - 1$	SS_M	$SS_M/(m - 1)$
Females (susceptible)	$f - 1$	SS_F	$SS_F/(f - 1)$
Males x Females	$(m - 1)(f - 1)$	SS_{MF}	$SS_{MF}/(m - 1)(f - 1)$
Error	$(mf - 1)(r - 1)$	SS_E	$SS_E/(mf - 1)(r - 1)$
Total	$rmf - 1$		

Table 4. Expected mean squares generated from the NC2 design.

Sources	df	EMS
Replications	r - 1	
Males	m - 1	$\sigma_e^2 + r\sigma_{mf}^2 + rf\sigma_m^2$
Females	f - 1	$\sigma_e^2 + r\sigma_{mf}^2 + rm\sigma_f^2$
Males x Females	(m - 1)(f - 1)	$\sigma_e^2 + r\sigma_{mf}^2$
Error	(mf - 1)(r - 1)	σ_e^2

$$\sigma^2_m = Cov_{P(HS)} = \frac{(1+F_m)}{4}\sigma^2_A + \frac{(1+F_m)^2}{16}\sigma^2_{AA} + \ldots$$

where σ^2_m is the variance of the random effects of male parents.

$Cov_{P(HS)}$ is the covariance of half-sibs (male in common).

F_m is the inbreeding coefficient for the male parents.

$$\sigma^2_f = Cov_{M(HS)} = \frac{(1+F_f)}{4}\sigma^2_A + \frac{1+F_f}{4}^2\sigma^2_{AA} + \ldots$$

where σ^2_f is the variance of the random effects of the female parents.

F_f is the inbreeding coefficient for the female parents.

$Cov_{M(HS)}$ is the covariance of half-sibs (female in common).

$$\sigma^2_{mf} = Cov_{(FS)} - Cov_{P(HS)} - Cov_{M(HS)}$$

where σ^2_{mf} is the variance of male x female interaction effects.

$$\sigma^2_{mf} = \frac{(1+F_m)(1+F_f)}{4}\sigma^2_D + \frac{(1+F_m)(1+F_f)}{8}\sigma^2_{AA} +$$

$$\frac{(2+F_m+F_f)(1+F_m)(1+F_f)}{16}\sigma^2_{AD} + \frac{(1+F_m)^2(1+F_f)^2}{16}\sigma^2_{DD}$$

where σ^2_A is the additive variance.

σ^2_D is the dominance variance.

σ^2_{AA} is the additive x additive variance.

Assuming that the parents are completely inbred ($F_m = F_f = 1$) the above formulae become:

$$\sigma^2_m = \sigma^2_f = Cov_{(HS)} = \frac{1}{2}\sigma^2_A + \frac{1}{4}\sigma^2_{AA} + \ldots \quad (8)$$

$$\sigma^2_{mf} = Cov_{(FS)} - Cov_{P(HS)} - Cov_{M(HS)}$$

$$= \sigma^2_D + \frac{1}{2}\sigma^2_{AA} + \ldots \quad (9)$$

In the absence of epistasis, equation 8 can be written as:

$$\sigma^2_m = \sigma^2_f = Cov_{(HS)} = \frac{1}{2}\sigma^2_A \quad (10)$$

and equation 9 becomes:

$$\sigma^2_{mf} = Cov_{(FS)} - Cov_{P(HS)} - Cov_{M(HS)}$$

$$= \sigma^2_D \quad (11)$$

Test for goodness of fit. The chi-square test is a useful method for testing goodness of fit of Mendelian ratios (Hayes and Immer, 1972). The comparison between the observed and expected values is made by calculating the χ^2 statistic (Snedecor and Cochran, 1978; Steel and Torrie, 1980).

$$\chi^2 = \sum_{i=1}^{k} \frac{(O_i - e_i)^2}{e_i} \qquad (12)$$

where O_i and e_i are the corresponding observed and expected values, in a set which consists of k pairs of such values.

The degree of freedom is one less than the number of cells. The distribution of χ^2 approximates the theoretical χ^2 distribution with k-1 degrees of freedom (Horn, 1977). It was also pointed out that this approximation is not a good one if the expected cell frequencies determined by the hypothesized distribution (not necessarily the observed cell frequencies) are small. In this case, the χ^2 statistic tends to be larger than the theoretical chi-square it is supposed to estimate.

Steel and Torrie (1980), in their book, provide the regions of acceptance for the various genetic ratios together with the probabilities of making a wrong decision.

Estimation of heritability. The term "heritability" evokes the image of transmission of characters from parents to offspring. It is used in biology to characterize the resemblance of related individuals in terms of a given characteristic, and to analyze the genetic and environmental causes of this resemblance.

Broad sense concept of heritability measures the degree to which the phenotype of an individual is genetically determined, while heritability in the narrow sense measures the degree to which the phenotype is passed on to offspring (under random mating).

Jacquard (1983) defined heritability in the narrow sense of a character in a population as the slope of the linear regression line (if it exists) of the measurements of the character amongst children of the mean of the measurements of the character for their two parents.

The standard regression model of Y on X is:

$$Y_i = a + bX_i + e_i \tag{13}$$

where Y_i is the measurement of character of offsprings.

X_i is the measurement of character of parental plant.

e_i is the error associated with Y_i.

The slope is given by the following formula:

$$b = \frac{\Sigma xy}{\Sigma x^2} = \frac{\Sigma(X_i - \bar{X})(Y_i - \bar{Y})}{\Sigma(X_i - \bar{X})^2} = \frac{\sigma^2_{xy}}{\sigma^2_x}$$

where σ_{xy} is the covariance of parent-offspring.

σ^2_x is the variance of the measurements on the parental plant.

But $$\sigma_{xy} = \frac{1}{2}\sigma^2_A + \frac{1}{4}\sigma^2_{AA} + \frac{1}{8}\sigma^2_{AAA}$$

Then, assuming no epistasis,

$$b = \frac{1}{2}(\sigma^2_A/\sigma^2_X) = \frac{1}{2}(\sigma^2_A/\sigma^2_X) \tag{14}$$

The two most commonly used regression estimators appear to be (Smith and Kinman, 1965):

$$2b = h^2 \tag{15}$$

$$b = h^2 \tag{16}$$

The first is an appropriate estimator for the regression of offspring on parent in a bisexual population when the parent is non-inbred. The second is appropriate for estimating heritability using selfed progeny if the parent is noninbred. However, this second estimator will overestimate heritability if the inbreeding coefficient is greater than zero or if there is dominance. Inbreeding will affect both estimators through σ_x^2. Equation 16 is also appropriate if you are regressing progeny on the midparent value. Using the notation of Smith and Kinman (1965), the slope b can be written as follows for selfed progeny:

$$b = 2r_{XY}(\sigma_A^2/\sigma_P^2) \tag{17}$$

where σ_p^2 is the total variance affected by inbreeding.

r_{XY} equals $\frac{1}{2}(1 + F_X)$.

F_X is the coefficient of inbreeding of the parent.

Thus $$b/2r_{XY} = \sigma_A^2/\sigma_P^2 = h^2 \tag{18}$$

This is the estimate of heritability in the narrow sense. For the specific case of the regression of F_3 offspring on F_2 parents, Luciano et al. (1965) give the estimate of heritability using equation 17:

$$b = 2r_{F_3,F_2}(\sigma_A^2/\sigma_P^2) \tag{19}$$

where r_{F_3,F_2} equals $\frac{1}{2}(1 + 0.5) = \frac{3}{4}$

Thus, when $F = \frac{1}{2}$, equation 19 becomes:

$$b = \frac{3}{2}(\sigma_A^2/\sigma_P^2) = \frac{3}{2} h^2$$

$$h^2 = \frac{2}{3} b \tag{20}$$

In the opinion of Cahaner and Hillel (1980), the correlations between parents and progeny means are usually used to estimate the heritability. Smith and Kinman (1965) give the estimate of heritability by using the correlation coefficient $r_{F3,F2}$ between the traits of F_2 generation and the mean of the traits in the F_3 generation. The heritability is expressed by the following relationship:

$$h^2 = \frac{3}{4} r_{F_3,F_2} \tag{21}$$

RESULTS AND DISCUSSION

In the fall of 1982 an attempt was made to produce a diallel set of crosses between long grain varieties and pure lines which differed in their resistance to sheath blight. Crosses were made in the greenhouse and, apparently due to difficulty with satisfactory pollen production and seed set, some of the crosses failed to produce seed. Crosses which were successful were listed in Table 2. F_1 plants from these crosses produced a satisfactory amount of F_2 seed during the summer of 1983. The remaining F_1 seeds did not germinate and therefore F_1 plants were not included in the test.

Frequency Distribution of Parental Lines and F_2 Populations

Parental lines and F_2 progeny populations were evaluated for sheath blight reaction in the greenhouse in the fall of 1983. The mean ratings of parental lines and F_2 populations from this test are shown in Table 5. Infection appeared to be present at a more or less uniform level throughout all flats in this test.

In the test the seedlings remained in the humidity chamber for two weeks following inoculation and the mean rating, on a 0-9 scale, of the susceptible parent lines P_1, P_2, P_3 and P_4 ranged from 6.9 to 7.8 and the mean rating of the resistant lines P_5, P_6 and P_7 ranged from 3.1 to 3.9. All the susceptible parental lines had a modal class of 8 and ratings of individual plants ranged from 5 to 9 (Figs. 1-4). The modal class of the resistant parents was 3 or 4

Table 5. Mean reaction to sheath blight and population size of parental lines and F_2 populations, inoculated and placed in the humidity chamber for two and four weeks.

Cross	Two Weeks in Humidity Chamber		Four Weeks in Humidity Chamber	
	Means Over Four Replications	Population Size	Means Over Four Replications	Population Size
P_1	7.6[1]	52	6.7[1]	65
P_2	7.8	73	7.0	80
P_3	6.9	60	7.2	77
P_4	7.1	76	6.6	45
P_5	3.1	61	3.8	80
P_6	3.9	75	4.5	54
P_7	3.4	72	3.6	80
F_2 Progeny				
$P_1 \times P_2$	7.2	217	6.9	240
$P_2 \times P_5$	5.1	224	5.5	240
$P_5 \times P_2$	4.8	238	5.9	80

Table 5, continued

Cross	Two Weeks in Humidity Chamber		Four Weeks in Humidity Chamber	
	Means Over Four Replications	Population Size	Means Over Four Replications	Population Size
$P_1 \times P_6$	5.7	202	5.8	239
$P_1 \times P_7$	5.8	290	--	--
$P_2 \times P_6$	5.5	189	6.3	145
$P_2 \times P_7$	4.8	235	--	--
$P_3 \times P_6$	5.2	199	5.3	240
$P_3 \times P_7$	5.7	76	5.5	239
$P_4 \times P_6$	5.6	229	5.5	240
$P_4 \times P_7$	5.1	212	5.4	237

[1]Individual seedlings were rated on a 0-9 scale, 0 = immune, 9 = very severe infection.

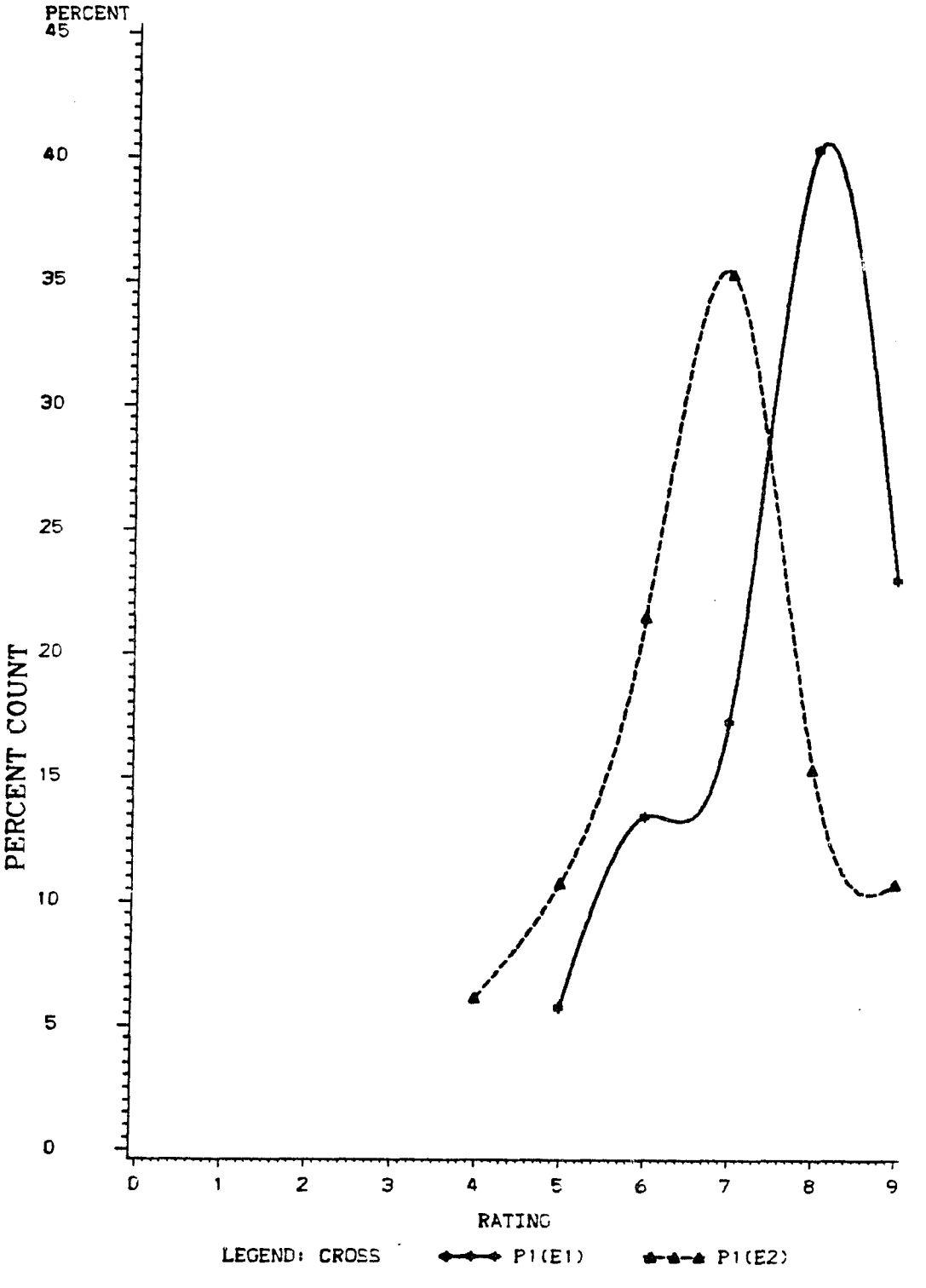

Figure 1. Frequency distribution of parental line P1 inoculated for 2 and 4 weeks.
P1=Lebonnet, E1=2 weeks , E2=4 weeks.

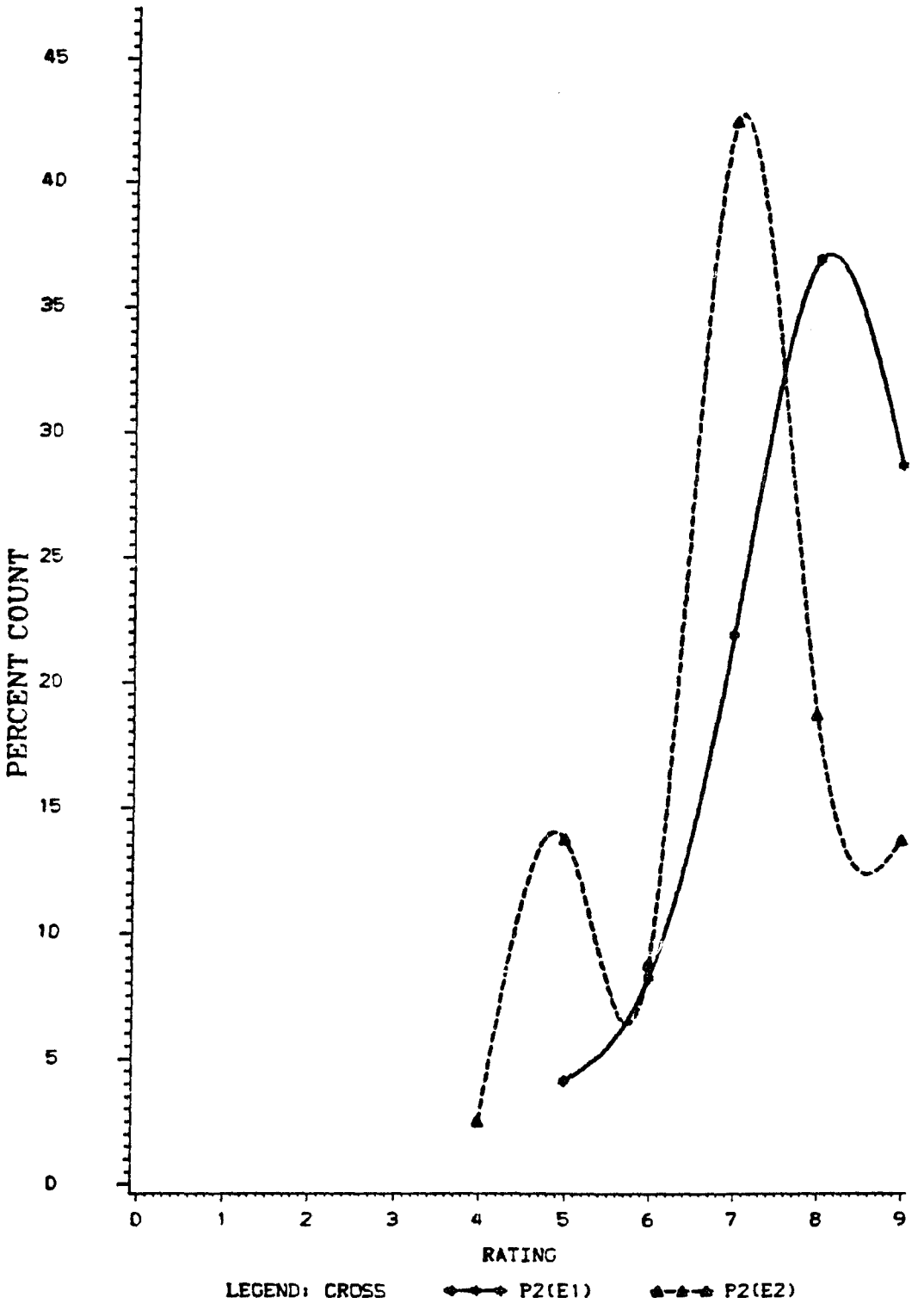

Figure 2. Frequency distribution of parental line P2 inoculated for 2 and 4 weeks.
P2=Labelle , E1=2 weeks , E2=4 weeks.

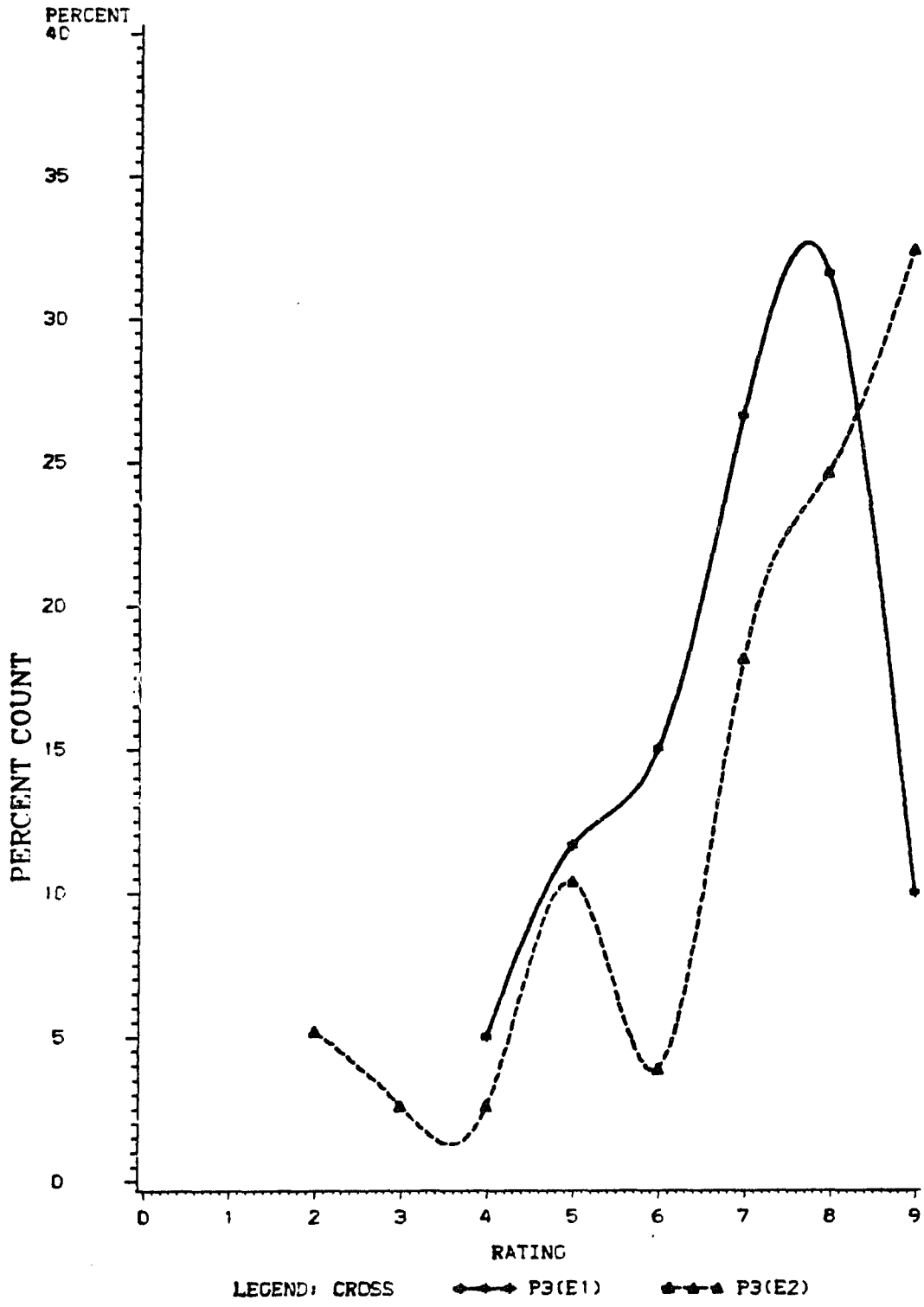

Figure 3. Frequency distribution of parental line P3 inoculated for 2 and 4 weeks.
P3=Starbonnet,E1=2 weeks , E2=4 weeks.

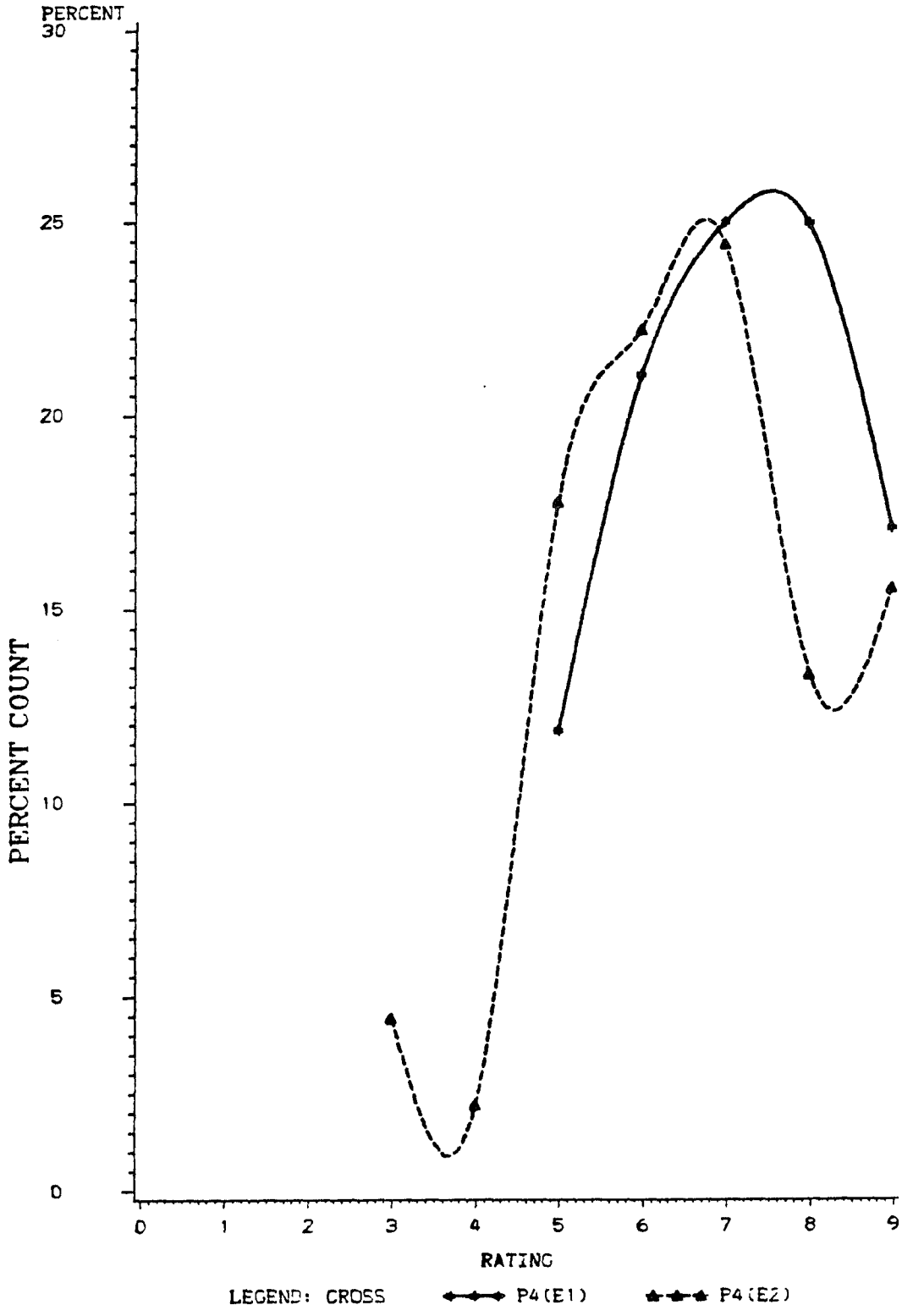

Figure 4. Frequency distribution of parental line P4 inoculated for 2 and 4 weeks.
P4=Leah, E1=2 weeks , E2=4 weeks.

and ratings of individual plants of the lines ranged from 1 to 5. These results indicate that the lines rated resistant or susceptible under field conditions were also rated similarly in this test. The resistant parents received a rating of 2 under field conditions but the mean rating of these lines in this test was higher, 3.1 to 3.9, indicating the conditions in the test were more suitable for disease development than usually encountered under field conditions.

In the second test where the plants remained in the humidity chamber for four weeks, the results were slightly affected by the length of time in the humidity chamber. The mean rating of the susceptible parent lines P_1, P_2, P_3 and P_4 ranged from 6.6 to 7.2 and the mean rating of the resistant lines ranged from 3.6 to 4.5 (Table 5). There was a slight decrease in mean rating of the susceptible lines P_1, P_2 and P_4 and a slight increase in mean rating over the susceptible parent P_3 and all the susceptible lines--P_1, P_2 and P_4--had a modal class of 7 and ratings of individual plants ranged from 4 to 9 in P_1 and P_2 and from 3 to 9 in P_4 (Figs. 1, 2 and 4). P_3 had a very wide range of ratings varying from 2 to 9 with a modal class of 9 (Fig. 3). The decrease in mean rating and the increase in range of the susceptible parent lines is probably due to the late application of inoculum to the plants. The inoculum was applied late because the first inoculum prepared was contaminated and discarded. By the time a good inoculum was obtained, the plants were 50 days old, and probably were at the culm elongation phase allowing the plants, to some extent, to outgrow the infection.

The ratings of individual plants of resistant parent lines inoculated and placed in the humidity chamber for four weeks ranged from 2 to 5, 3 to 6 and 2 to 6, respectively in P_5, P_6 and P_7 (Figs. 5-7). The increase in mean ratings of the resistant parent lines indicates that more plants shifted from moderately resistant to moderately susceptible. This suggests that the resistance of the parental lines tended to break down under conditions very favorable for the disease.

The F_2 progeny of the cross $P_1 \times P_2$ (susceptible x susceptible) had a disease reaction varying from 4 to 9 when the plants were inoculated and placed in the humidity chamber for two weeks. The frequency distribution of the F_2 progeny of the cross $P_1 \times P_2$ is close to that of the parents (Fig. 8). This suggests that P_1 and P_2 parental lines have the same genes affecting resistance and susceptibility to sheath blight. The ratings of individual plants of the F_2 progeny of the cross $P_1 \times P_2$ when the plants were inoculated and placed in the humidity chamber for four weeks ranged from 2 to 9 with a modal class of 8. Except for a few plants which were rated 3 and 4, the frequency distribution of the F_2 progeny appeared similar to that of both parents thus supporting the contention that the P_1 and P_2 lines have genetically similar susceptibility to sheath blight.

Since the P_1 and P_2 parents appear to be genetically similar parents susceptible to sheath blight, the results obtained from the progeny of crosses of the two lines and the resistant sister selections P_6 and P_7 will be discussed as a group. The mean rating

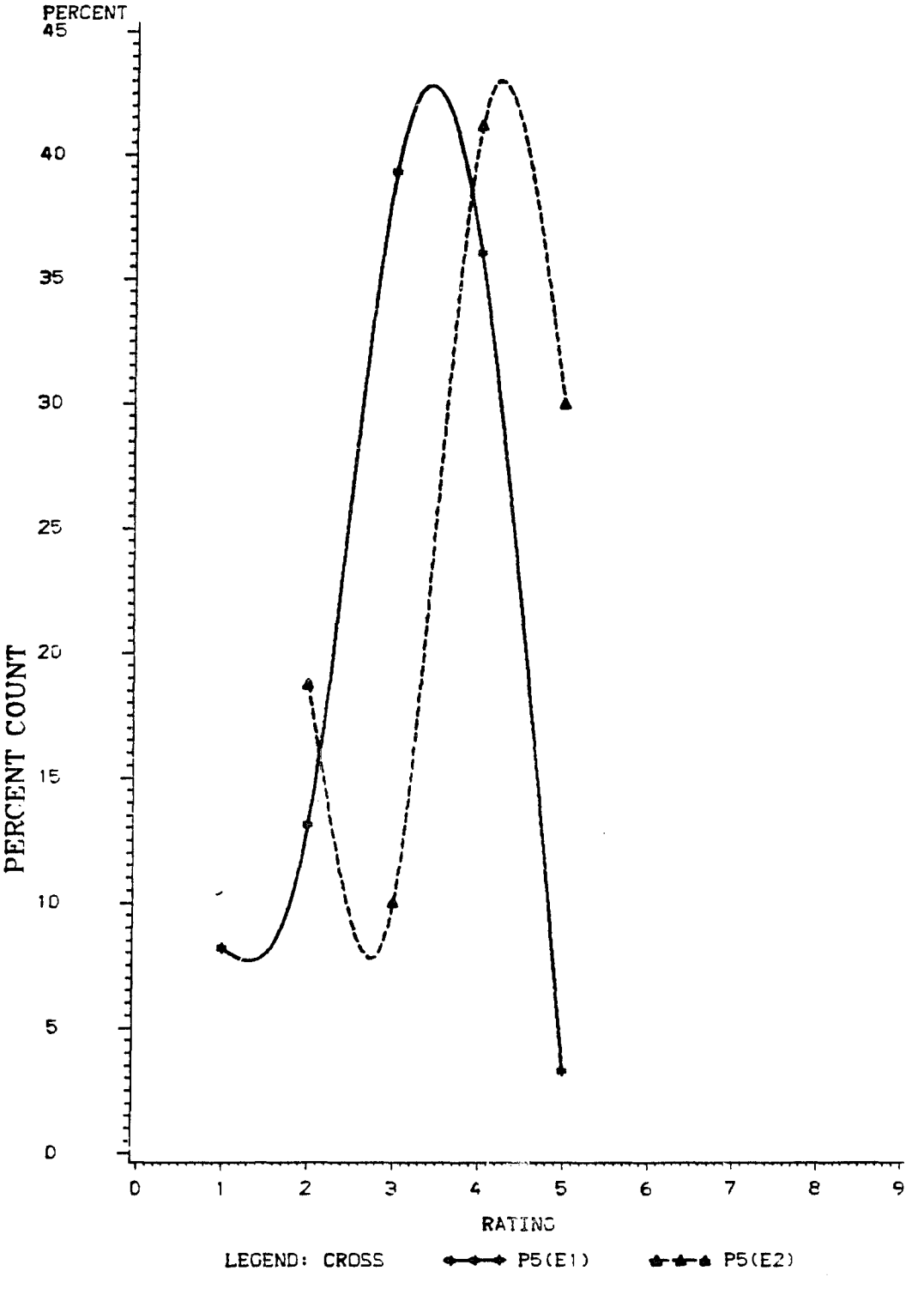

Figure 5. Frequency distribution of parental line P5 inoculated for 2 and 4 weeks.
P5=L201 , E1=2 weeks , E2=4 weeks.

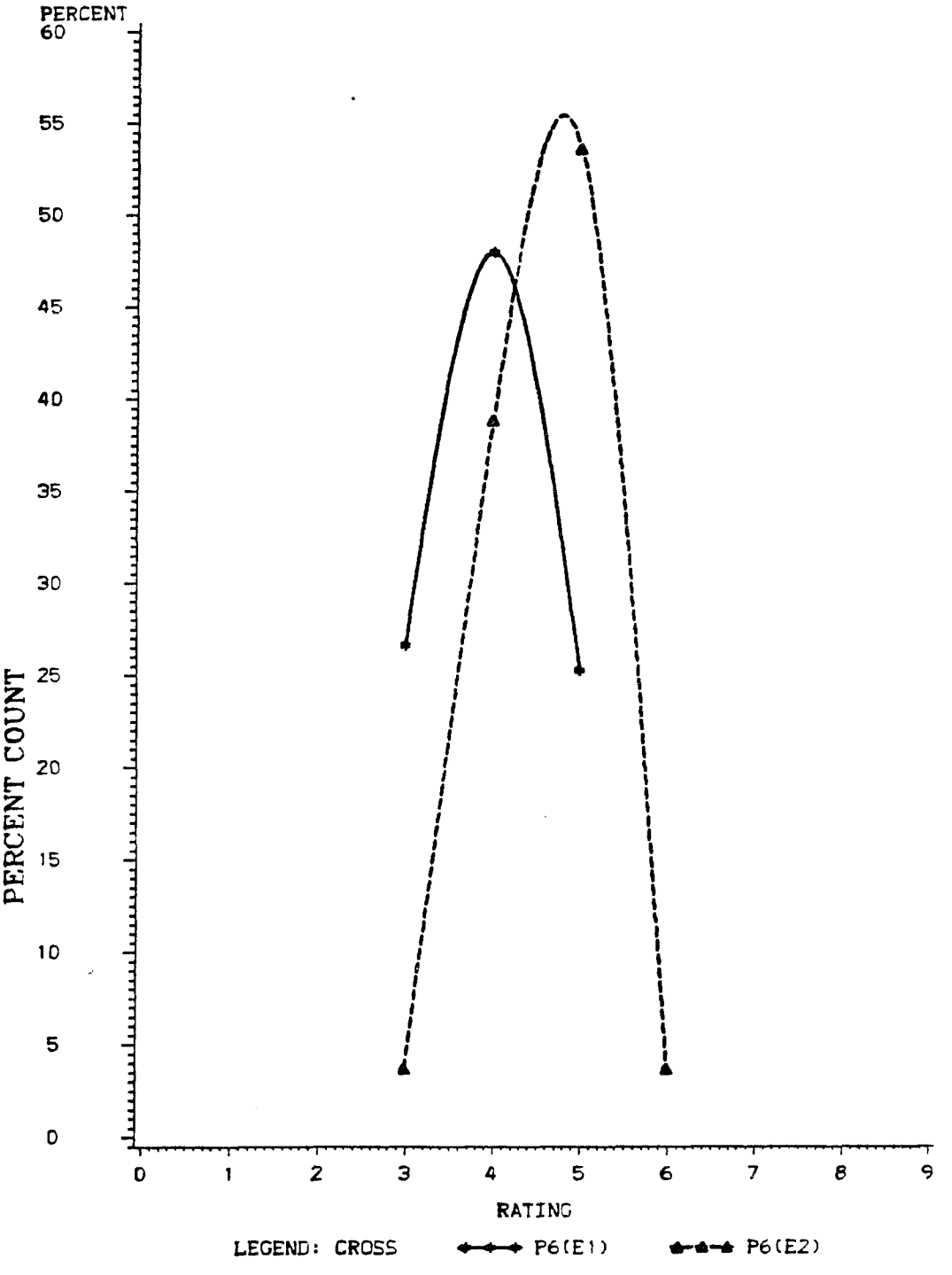

Figure 6. Frequency distribution of parental line P6 inoculated for 2 and 4 weeks.
P6=RU7902185 , E1=2 weeks , E2=4 weeks.

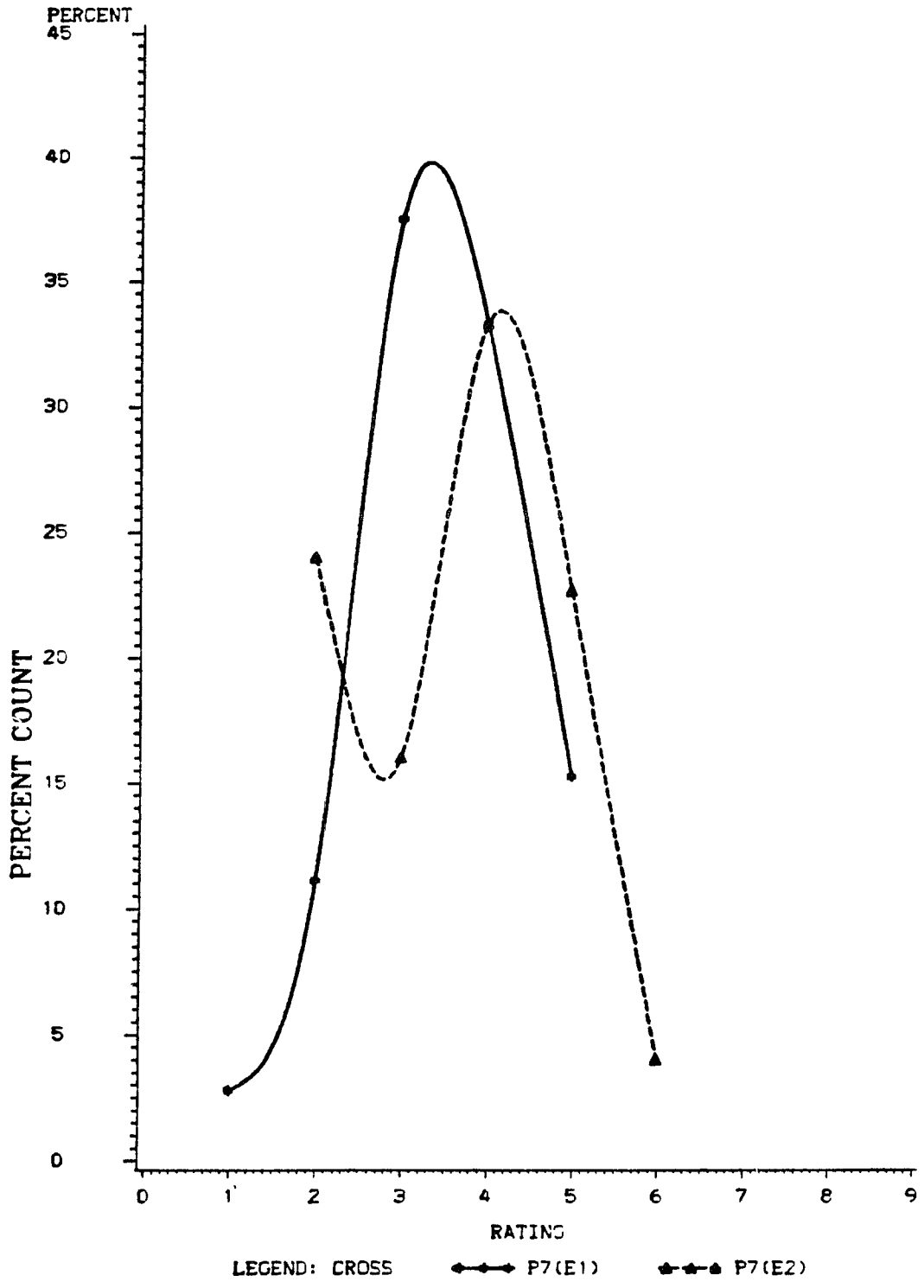

Figure 7. Frequency distribution of parental line P7 inoculated for 2 and 4 weeks.
P7=RU7902191 , E1=2 weeks , E2=4 weeks.

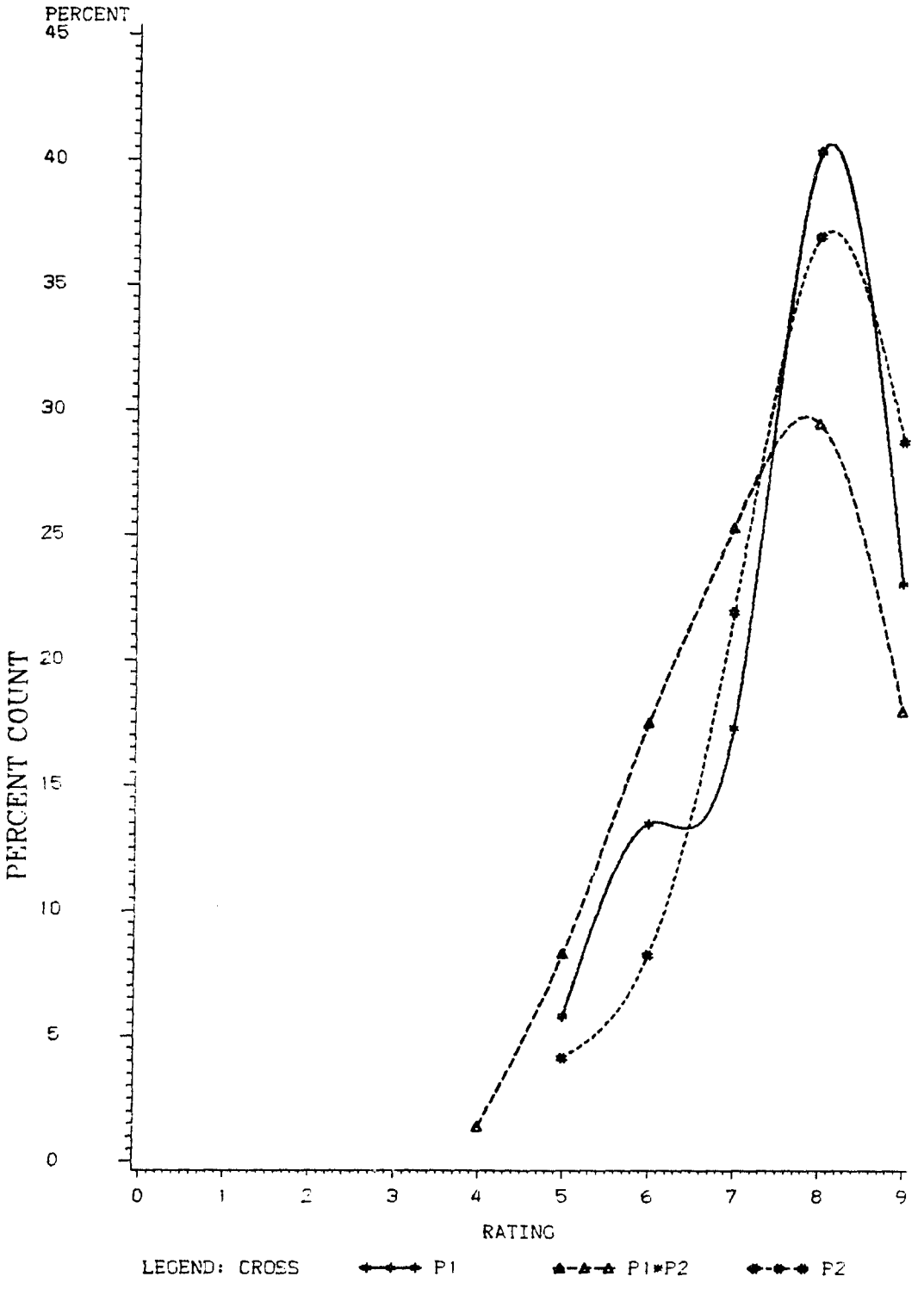

Figure 8. Frequency distribution of parental lines and F2 generation of the cross P1*P2 inoculated for 2 weeks. P1=Lebonnet, P2=Labelle.

of the F_2 progeny of the $P_1 \times P_6$, $P_1 \times P_7$ and $P_2 \times P_6$ crosses were similar--5.7, 5.8 and 5.5, respectively in the test which remained in the humidity chamber for two weeks. The mean of the $P_2 \times P_7$ F_2 progeny was 4.8 which was approximately or closely lower than the other progeny in this group. The frequency distribution of rating classes of these for F_2 progeny (Figs. 9, 10, 11 and 12) were similar, showing a bimodal distribution with modes at 5 and 7 except for $P_2 \times P_7$ F_2 progeny with a mode at 4 for the resistant group. Based on this bimodal distribution of the F_2 progeny and the rating distribution for the homozygous parent the F_2 populations were divided into resistant and susceptible groups with the groups being divided between the 5 and 6 rating classes so that plants with 5 or less were considered resistant and plants rated 6 or higher were considered to be susceptible.

The estimation of a fit to classical two-class ratio was calculated and the results are presented in Tables 6 and 7. The F_2 progeny of the $P_1 \times P_6$ and $P_1 \times P_7$ crosses fit a 27:37 resistant to susceptible ratio and $P_2 \times P_7$ progeny fit a 3:1 resistant to susceptible ratio. If P_1 and P_2 are genetically the same for sheath blight resistance as previously indicated, the overall explanation apparently is that resistance in the P_6 and P_7 lines is controlled by complementary dominant or partially dominant genes at two independent loci which are not present in the susceptible P_1 and P_2 parents. The fit of the genetic ratio probably can be explained as

Table 6. Chi-square estimation of goodness of fit of the F_2 progeny of the genetic ratios (plants inoculated and placed in the humidity chamber for two weeks).

Cross	Ratio	Number of Observations R:S[1]	χ^2 Probability Level
$P_1 \times P_6$	3:1	95:107	<0.01
	9:7		<0.01
	27:37		0.10 - 0.25
$P_1 \times P_7$	3:1	98:111	<0.01
	9:7		<0.01
	27:37		0.10 - 0.25
$P_2 \times P_6$	3:1	99:90	<0.01
	9:7		0.25 - 0.50
	27:37		<0.01
$P_2 \times P_7$	3:1	163:72	0.025 - 0.050
	9:7		<0.01
	27:37		<0.01
$P_3 \times P_6$	3:1	122:77	<0.01
	9:7		0.10 - 0.25
	27:37		<0.01
$P_3 \times P_7$	3:1	36:40	<0.01
	9:7		<0.01
	27:37		0.25 - 0.50
$P_4 \times P_6$	3:1	117:112	<0.01
	9:7		0.10 - 0.25
	27:37		<0.01
$P_4 \times P_7$	3:1	127:85	<0.01
	9:7		0.25 - 0.50
	27:37		<0.01

Table 6, continued

Cross	Ratio	Number of Observations R:S[1]	χ^2 Probability Level
$P_2 \times P_5$	3:1	141:83	<0.01
	9:7		0.025 - 0.050
	27:37		<0.01
	27:37[2]	89:135	0.25 - 0.50
$P_5 \times P_2$	3:1	168:70	0.10 - 0.25
	9:7		<0.01
	27:37		<0.01
	27:37[2]	105:133	0.50 - 0.75

[1]R = 0-5 rating, S = 6-9 rating. Detailed data reported in Appendix Table 1.

[2]R = 0-4, S = 5-9 rating. Detailed data reported in Appendix Table 1.

Table 7. Chi-square estimation of goodness of fit of the F_2 progeny to the genetic ratios (plants inoculated and placed in the humidity chamber for four weeks).

Cross	Ratio	Number of Observations R:S[1]	χ^2 Probability Level
$P_1 \times P_6$	3:1	118:121	<0.01
	9:7		<0.01
	27:37		0.01 - 0.025
$P_2 \times P_6$	3:1	52:93	<0.01
	9:7		<0.01
	27:37		0.10 - 0.25
$P_3 \times P_6$	3:1	135:105	<0.01
	9:7		0.75 - 0.90
	27:37		<0.01
$P_3 \times P_7$	3:1	138:101	<0.01
	9:7		0.50 - 0.75
	27:37		<0.01
$P_4 \times P_6$	3:1	136:104	<0.01
	9:7		0.75 - 0.90
	27:37		<0.01
$P_4 \times P_7$	3:1	141:96	<0.01
	9:7		0.25 - 0.50
	27:37		<0.01
$P_2 \times P_5$	3:1	143:97	<0.01
	9:7		0.25 - 0.50
	27:37		<0.01
$P_5 \times P_2$	3:1	40:40	<0.01
	9:7		0.25 - 0.50
	27:37		0.10 - 0.25

[1]R = 0-5 rating, S = 6-9 rating. Detailed data reported in Appendix Table 2.

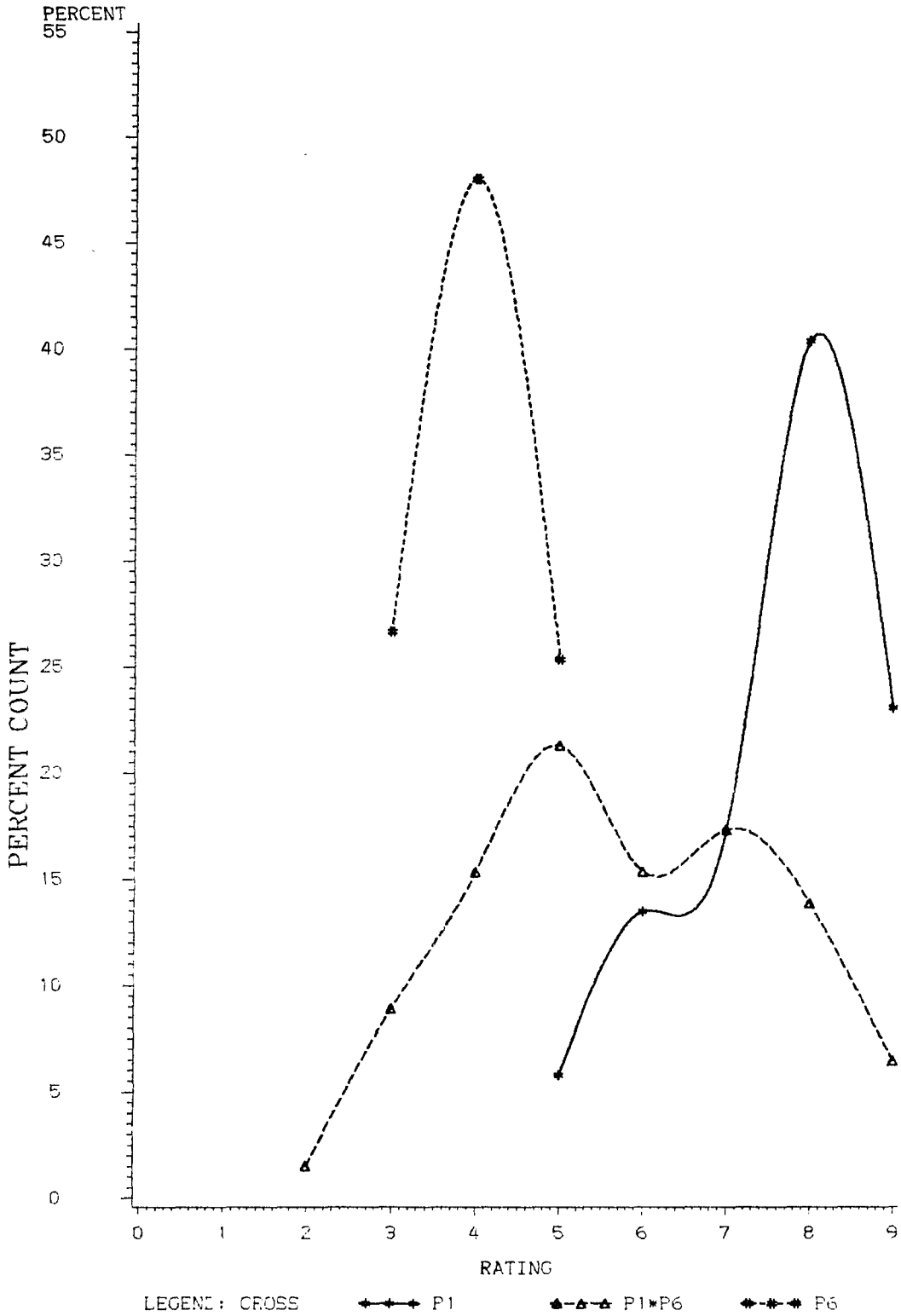

Figure 9. Frequency distribution of parental lines and F2 generation of the cross P1*P6 inoculated for 2 weeks. P1=Lebonnet, P6=RU7902185.

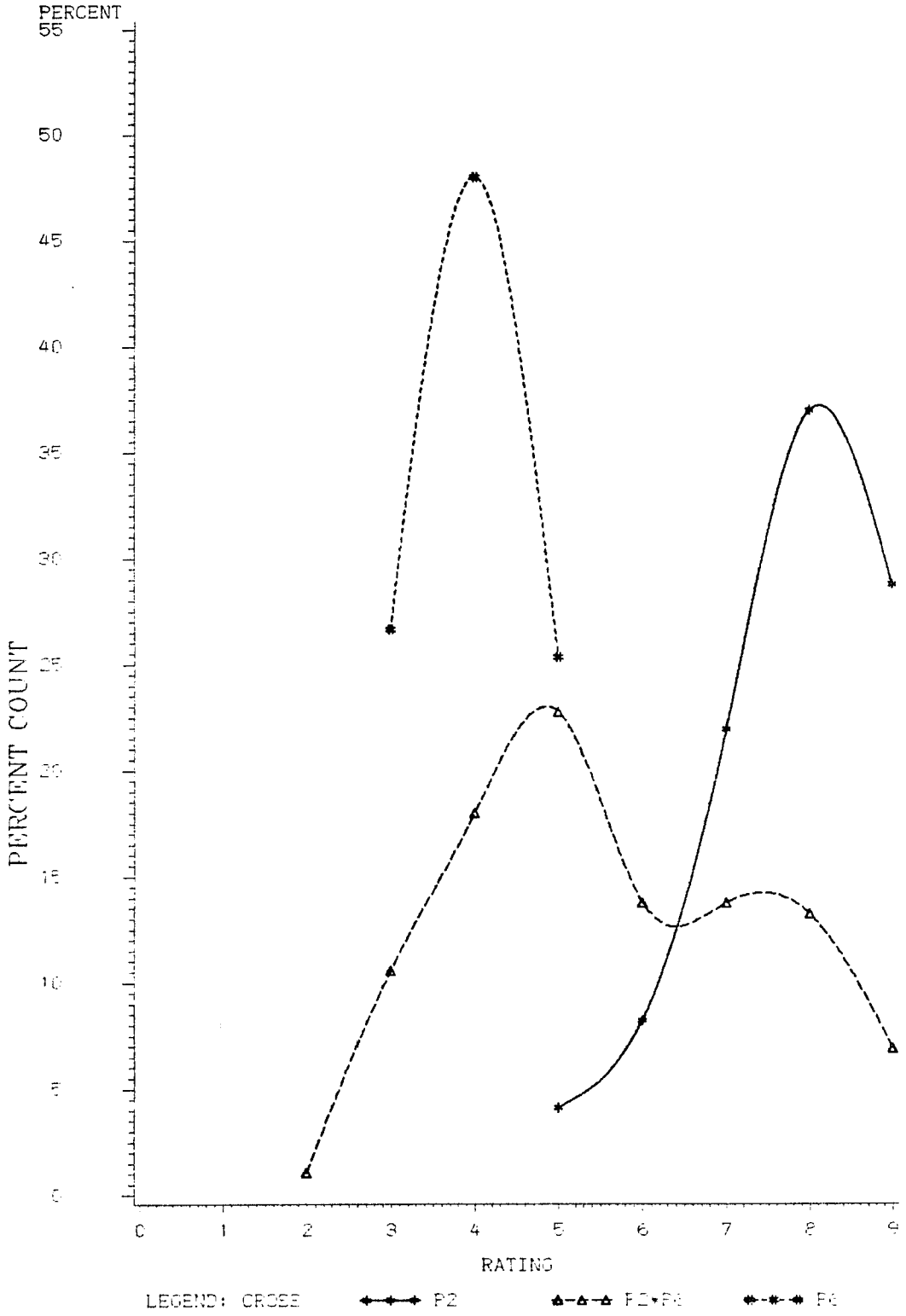

Figure 10. Frequency distribution of parental lines and F2 generation of the cross P2*P6 inoculated for 2 weeks. P2=Labelle, P6=RU7902185.

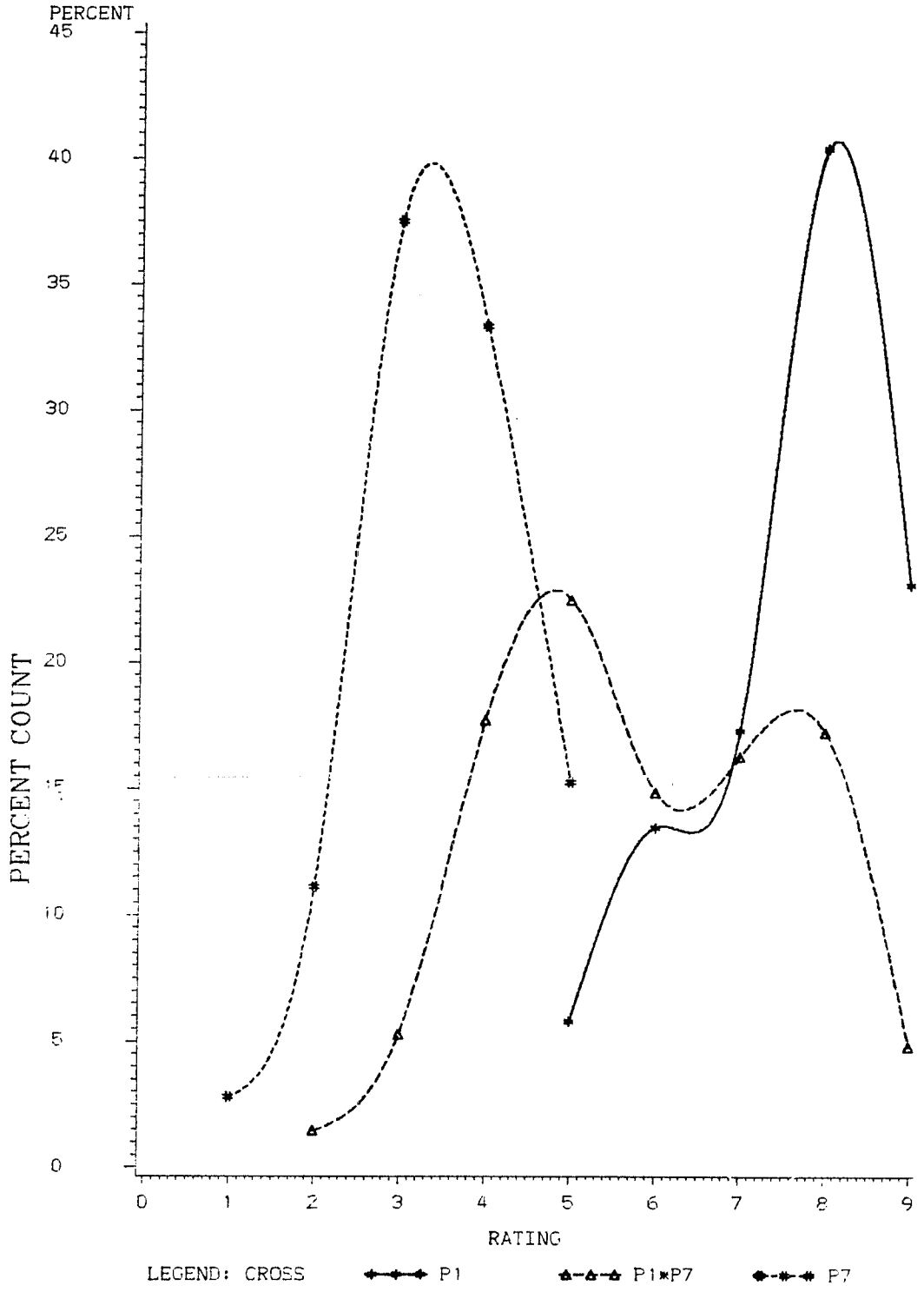

Figure 11. Frequency distribution of parental lines and F2 generation of the cross P1*P7 inoculated for 2 weeks. P1=Lebonnet, P7=RU7902191.

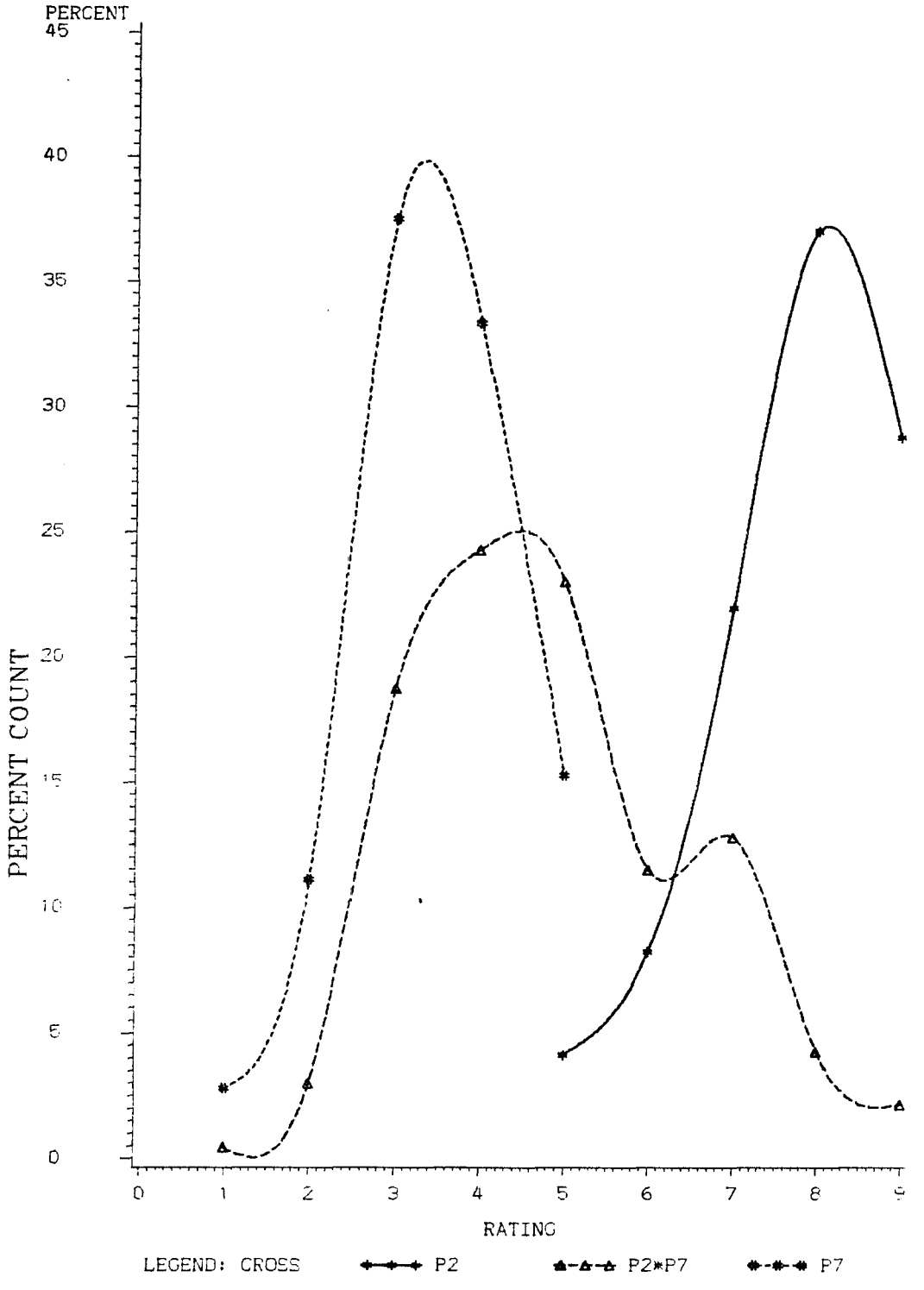

Figure 12. Frequency distribution of parental lines and F2 generation of the cross P2*P7 inoculated for 2 weeks. P2=Labelle, P7=RU7902191.

the result of a nonrandom misclassification of plants in the 5 and 6 rating classes which border the breakpoint between the resistant and susceptible groups. These misclassifications which may have occurred due to minor change(s) in disease infection could have had a large effect on the number of plants placed in either 5 or 6 rating classes. Since there were large numbers of plants in both the 5 and 6 rating classes, the possibility exists for major shifts in the resistant and susceptible groups due to relatively minor change in disease infection.

The F_2 progeny of the crosses involving the susceptible lines P_3 and P_4 ($P_3 \times P_6$, $P_3 \times P_7$, $P_4 \times P_6$ and $P_4 \times P_7$) had similar mean ratings which ranged from 5.1 to 5.6 when the plants remained in the humidity chamber for two weeks following inoculation. Individual plants were rated over a range of 1 to 9 in F_2 progeny crosses $P_3 \times P_6$ (Fig. 13) and $P_4 \times P_7$ (Fig. 16), 2 to 9 in F_2 progeny of $P_4 \times P_6$ (Fig. 15) and 3 to 9 in F_2 progeny of $P_3 \times P_7$ (Fig. 14). Two modal classes were observed at 5 and 7 in $P_3 \times P_6$ and $P_4 \times P_6$. The F_2 progeny of the cross $P_3 \times P_7$ had one mode at 5 and another between 7 and 8. The frequency distribution of the cross $P_4 \times P_7$ exhibits a 1:2:1 type of curve (Fig. 16). In the second test in which the plants were inoculated and placed in the humidity chamber for four weeks, the mean ratings of the four F_2 progeny did not differ. The individual ratings of the F_2 progeny of the crosses $P_3 \times P_6$ and $P_4 \times P_7$ ranged from 2 to 9 and that of the crosses $P_3 \times P_7$ and $P_4 \times P_6$ ranged from 3 to 9. Two modal classes at 4 and 7 were

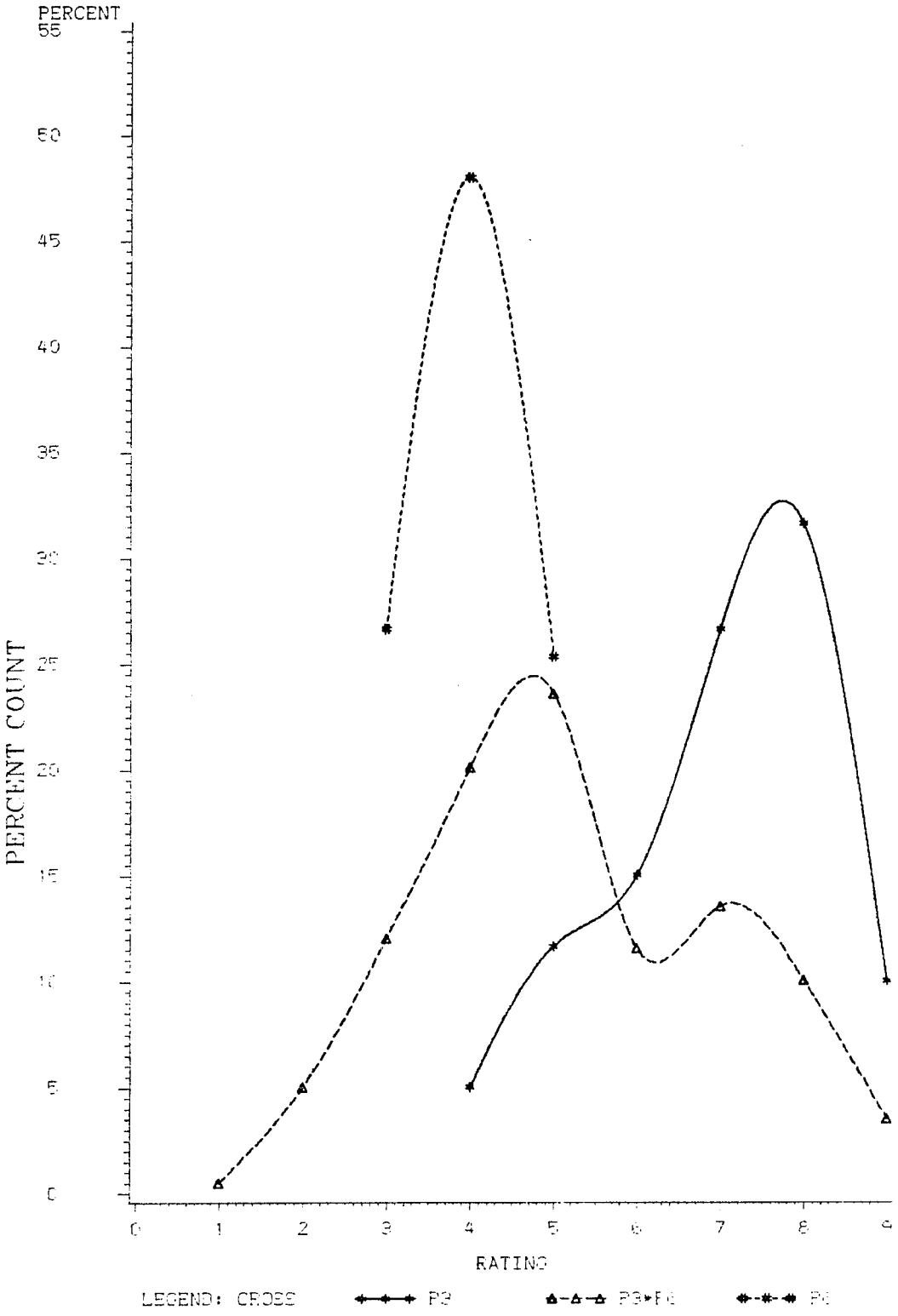

Figure 13. Frequency distribution of parental lines and F2 generation of the cross P3*P6 inoculated for 2 weeks. P3=Starbonnet, P6=RU7902185.

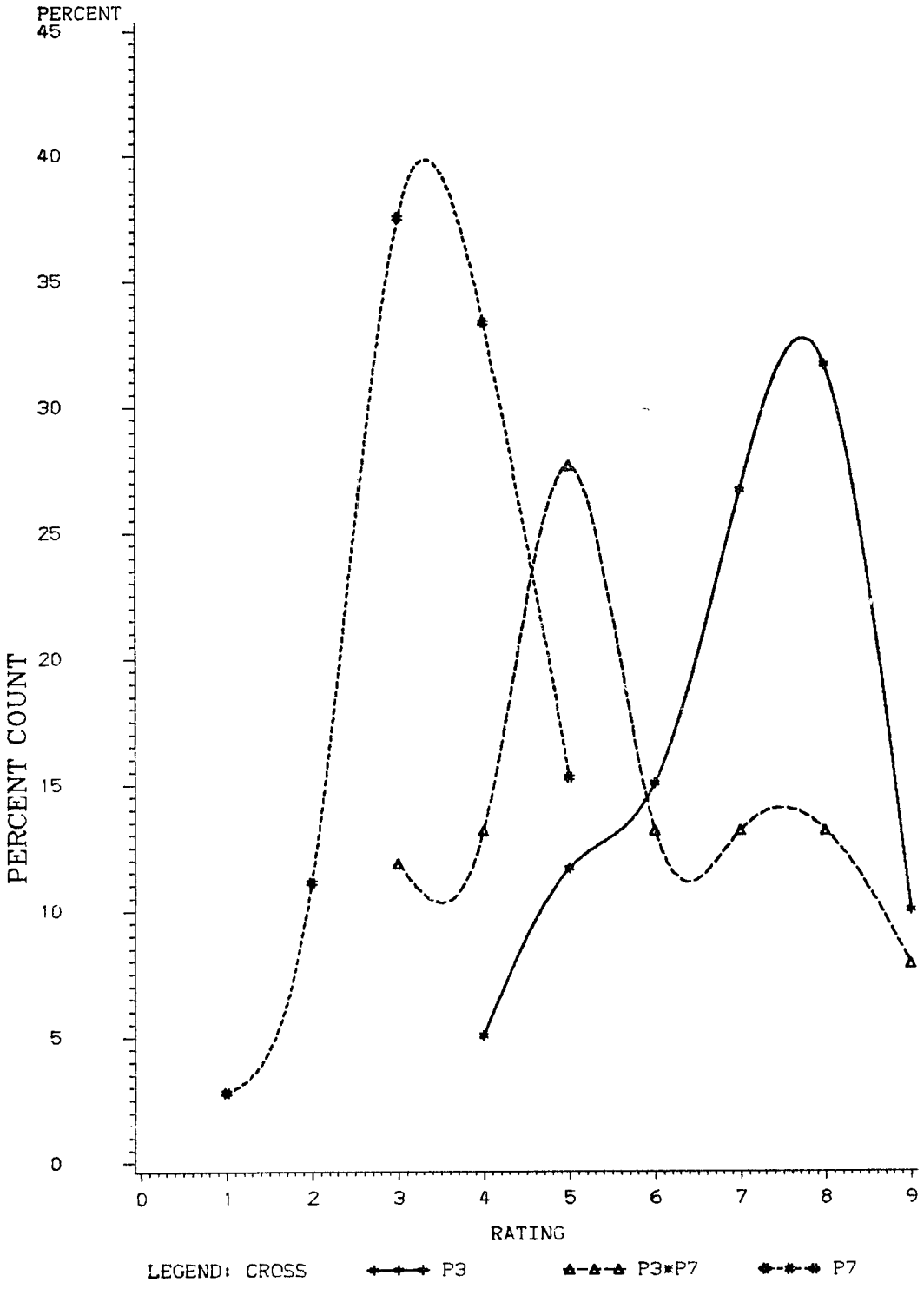

Figure 14. Frequency distribution of parental lines and F2 generation of the cross P3*P7 inoculated for 2 weeks. P3=Starbonnet, P7=RU7902191.

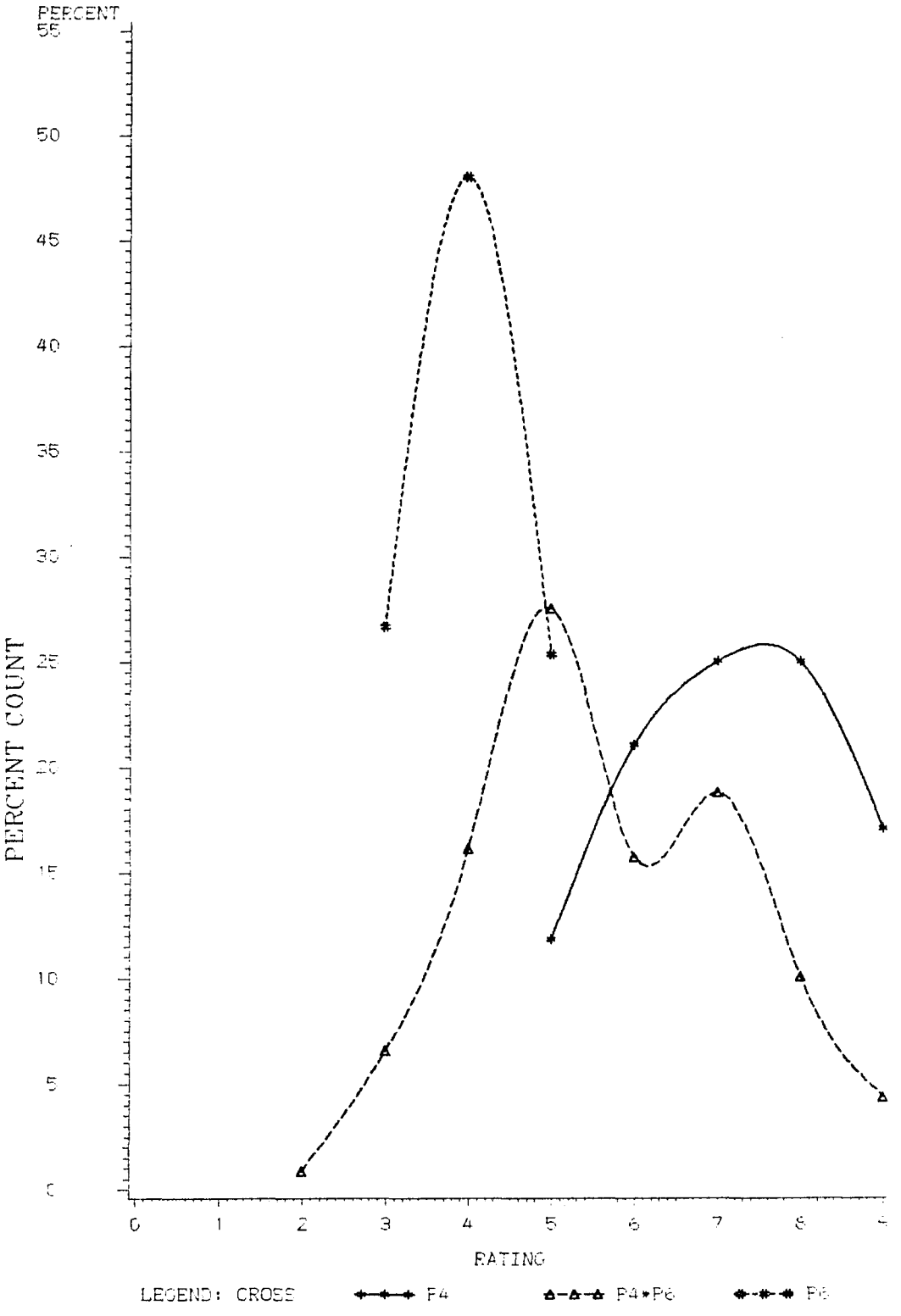

Figure 15. Frequency distribution of parental lines and F2 generation of the cross P4*P6 inoculated for 2 weeks. P4=Leah, P6=RU7902185.

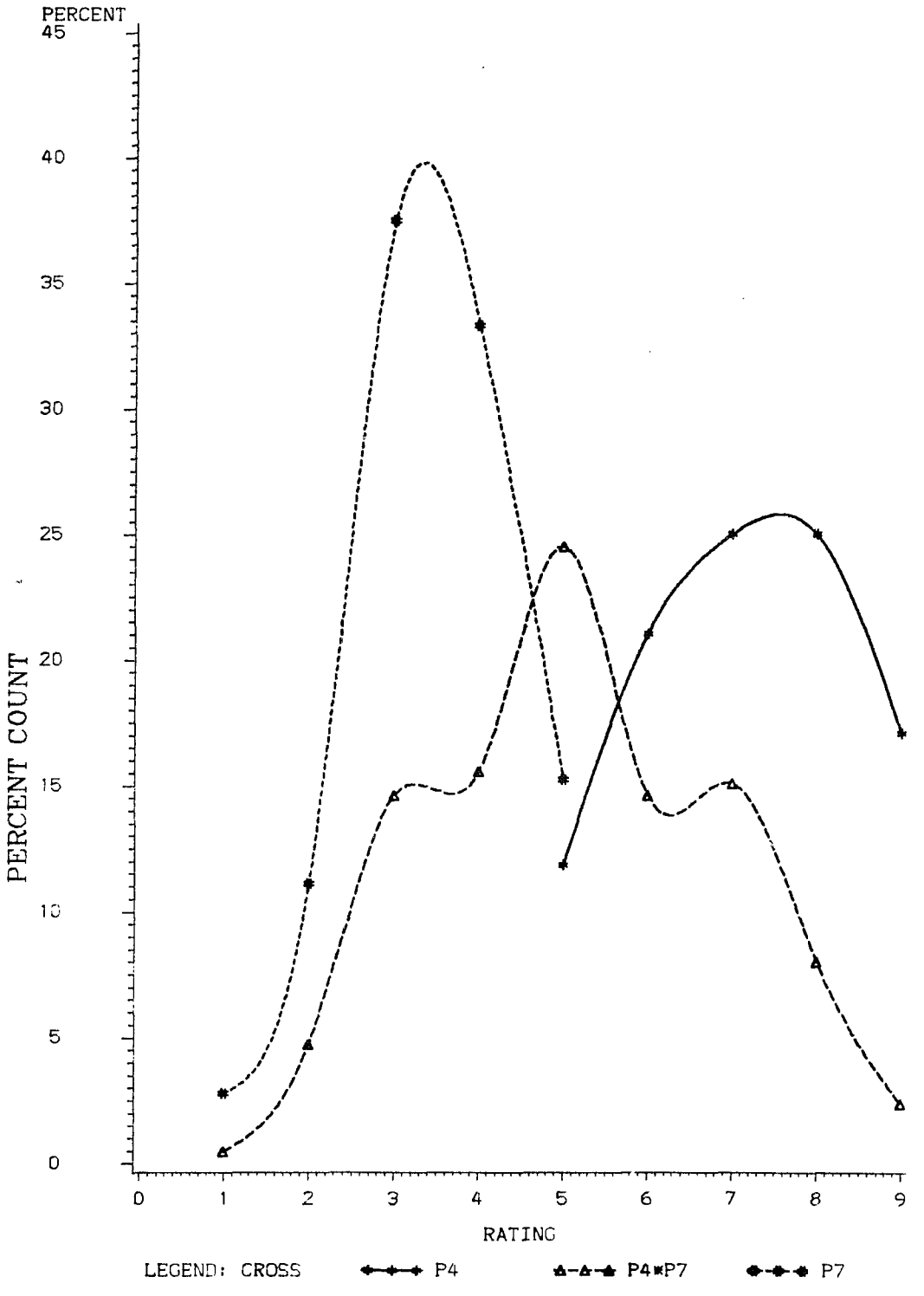

Figure 16. Frequency distribution of parental lines and F2 generation of the cross P4*P7 inoculated for 2 weeks. P4=Leah, P7=RU7902191.

observed in the crosses $P_3 \times P_6$, $P_4 \times P_6$ and $P_3 \times P_7$. The F_2 progeny of the cross $P_4 \times P_7$ had two modal classes at 5 and 7.

The estimation of goodness of fit of the F_2 progeny indicated that all the crosses except one fit the 9:7 ratio (Table 6), indicating that two pairs of complementary genes affect the resistance or susceptibility to sheath blight (Tables 6 and 7). The F_2 progeny of the cross $P_3 \times P_7$ did not fit a 9:7 ratio due to a deficiency of plants in the resistant group (Table 5). However it appears that this could be explained due to a misclassification of the plants in the 5 and 6 rating classes. A slight change in disease reaction or slight change in the judgement of individuals rating the plants could change a plant from a 5 to a 6 or a 6 to a 5. While this could be due to only a slight change in disease reaction this could have a substantial effect on the number of plants placed in the resistant or susceptible groups.

The F_2 progeny of the cross $P_2 \times P_5$ (susceptible x resistant) had a mean rating of 5.1 when the plants were inoculated and placed in the humidity chamber for two weeks. The ratings of individual plants ranged from 2 to 9 with modal classes 3, 5 and 7 (Fig. 17). The F_2 progeny of the reciprocal cross $P_5 \times P_2$ had a mean rating of 4.8 when the plants were inoculated and placed in the humidity chamber for two weeks. The rating of individual plants ranged from 2 to 9 with modal classes at 3, 5 and 7 (Fig. 18). The frequency distribution of the reciprocal crosses $P_2 \times P_5$ (Fig. 17) and $P_5 \times P_2$

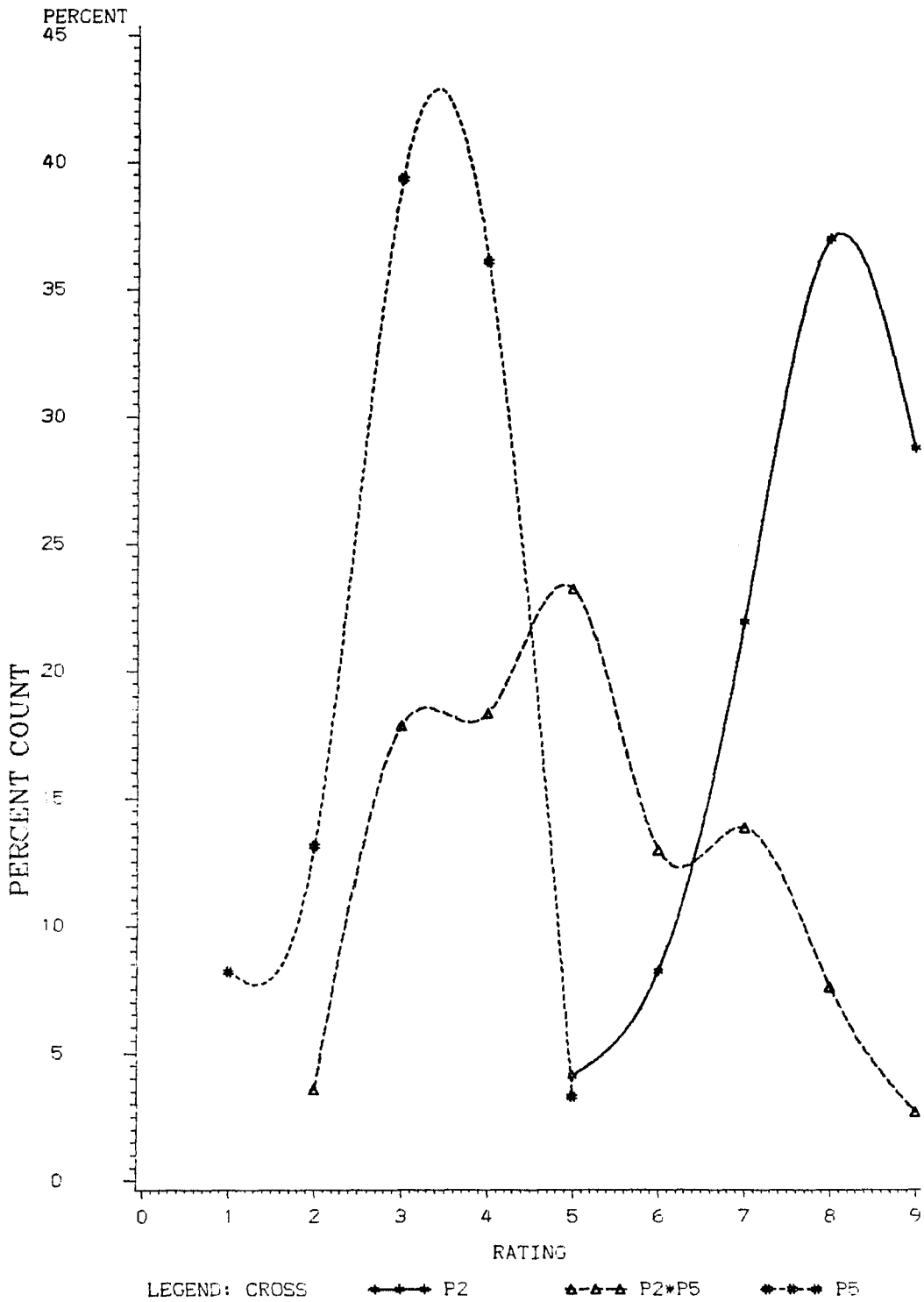

Figure 17. Frequency distribution of parental lines and F2 generation of the cross P2*P5 inoculated for 2 weeks. P2=Labelle, P5=L201.

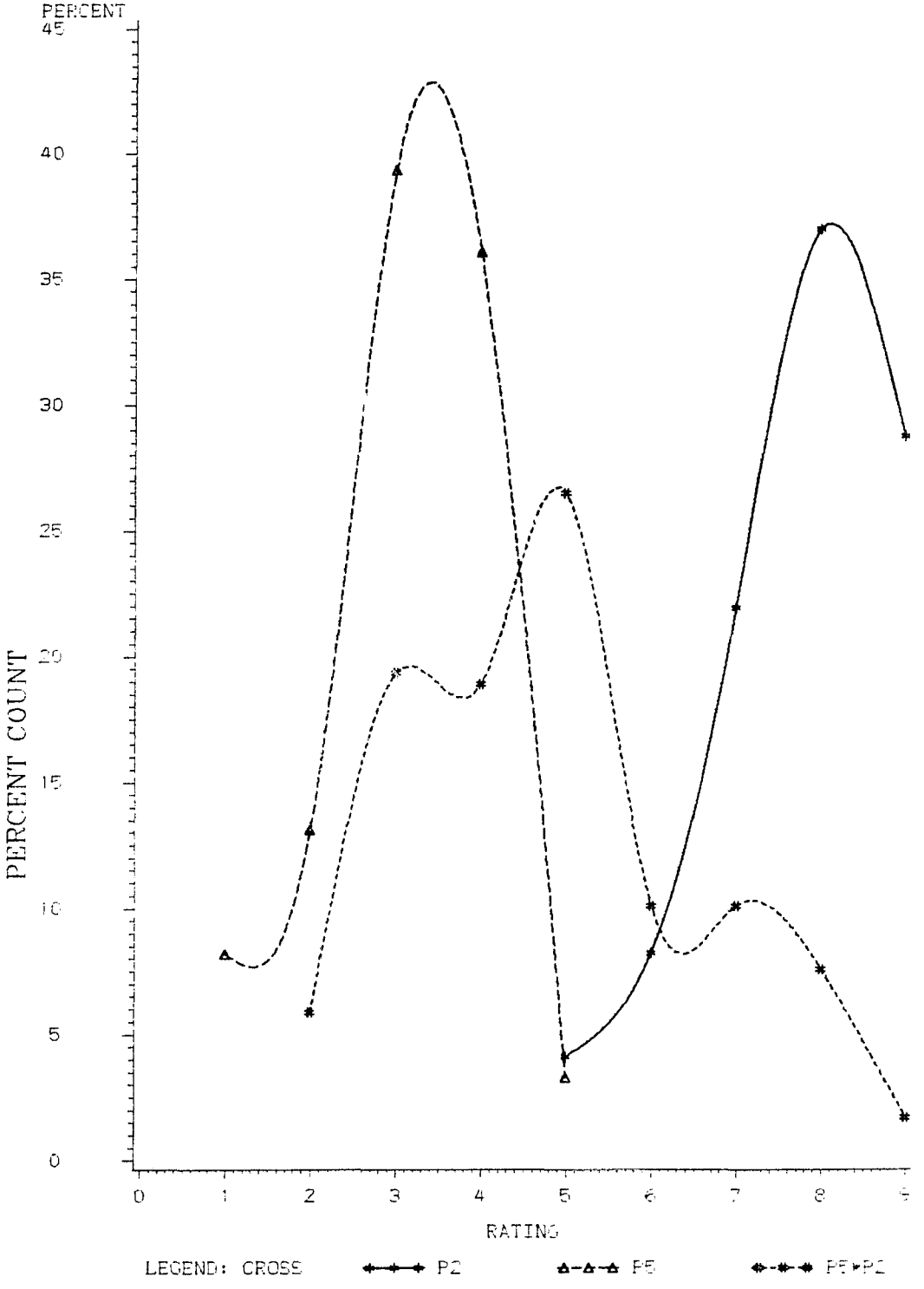

Figure 18. Frequency distribution of parental lines and F2 generation of the cross P5*P2 inoculated for 2 weeks. P5=L201, P2=Labelle.

(Fig. 18) was similar and the t-test showed no difference between the two populations, indicating an absence of reciprocal effects.

When the F_2 progeny of the $P_2 \times P_5$ and $P_5 \times P_2$ crosses were inoculated and placed in the humidity chamber for four weeks, the $P_2 \times P_5$ F_2 progeny had a mean of 5.5 with a range of individual plant ratings from 2 to 9 and two definite modal classes at 5 and 7. The $P_5 \times P_2$ F_2 progeny had a mean of 5.9 with a range of individual plant ratings from 4 to 9 and two definite modal classes at 5 and 7. There was no significant difference according to the t-test between the F_2 populations of the reciprocal crosses supporting the presence of no reciprocal cross effects.

Since the distribution of the F_2 progeny appeared to fall into two distinct groups with modes at 5 and 7, an estimation of goodness of fit for a two class genetic ratio for resistance and susceptible was calculated for these populations (Tables 6 and 7). Previous studies have indicated that resistance is dominant or partially dominant and since, the modal class for the resistant parent in the crosses appeared to be either 3 or 4. A modal class of 5 for the resistant group in the F_2 progeny suggests partial dominance for resistance in the crosses. Based on the rating estimation of the homozygous parent as well as the F_2 distribution, the break between the resistant and susceptible group appeared to be between the 5 and 6 rating classes. Therefore any plants classified as 5 or lower were considered resistant while plants rated 6 or higher were considered susceptible. Using this break point, the

resistant and susceptible groups were a good fit to a 9:7 ratio, P = 0.250 - 0.50, in the test which remained in the humidity chamber for four weeks. However, the ratio for the F_2 progeny in the test which remained in the humidity chamber for only two weeks did not fit either a 3:1 or a 9:7 ratio with the resistant class being too small for a 3:1 ratio and too large for a 9:7 ratio. It would appear that the best explanation for this is since the 5 and 6 ratings are border ratings that a slight non random misclassification of plants in the two classes could cause the deviation from the expected. For example, if a few plants which should have been rated as 6 were instead rated 5, the resistant classes could be inflated although only a small change in disease rating actually was present. It would appear that the best explanation for the results obtained is that the resistance in the P_5 (L201) parent was controlled by complementary dominant or partially dominant genes at two loci which were not present in the susceptible P_2 (Labelle) parent.

Data on height of infection as a percentage of total sheath height had a wide range over all parental lines (Table 8). The range reached 100% in all parents except P_5 which ranged from 9.0 to 92.5%. Data in Table 5 showed that susceptible lines ranged from 27 to 100% and resistant lines ranged from 7 to 100%. However, the range of the resistant line P_6 did not differ from that of the susceptible lines P_1 and P_2. Using individual plant data, a correlation coefficient of 0.439 was found between the disease ratings 0 to 9 and the height of infection as a percentage of total sheath height.

Table 8. Means and ranges of height of highest lesions converted to percent of total sheath height and disease ratings of parental lines and F_2 progeny over four replications.

Parental and F_2 Populations	Percent Sheath Blight Infection		Disease Rating (0-9)	
	Mean	Range	Mean	Range
P_1	84.4	43.3 - 100	7.6	5 - 9
P_2	83.1	48.4 - 100	7.8	5 - 9
P_3	78.3	33.3 - 100	6.9	4 - 9
P_4	76.9	27.0 - 100	7.1	5 - 9
P_5	53.7	9.0 - 92.5	3.1	1 - 5
P_6	74.4	45.5 - 100	3.9	3 - 5
P_7	63.0	7.6 - 100	3.4	1 - 5
$P_1 \times P_2$	85.4	25.9 - 100	7.2	4 - 9
$P_1 \times P_6$	77.4	27.1 - 100	5.7	2 - 9
$P_1 \times P_7$	72.3	10.7 - 100	5.8	2 - 9
$P_2 \times P_5$	65.9	13.3 - 100	5.1	2 - 9
$P_2 \times P_6$	71.7	6.6 - 100	5.5	2 - 9
$P_2 \times P_7$	67.0	7.5 - 100	4.8	1 - 9
$P_3 \times P_6$	75.7	9.3 - 100	5.2	1 - 9
$P_3 \times P_7$	69.4	40.6 - 100	5.7	3 - 9
$P_4 \times P_6$	73.1	13.7 - 100	5.6	2 - 9
$P_4 \times P_7$	66.8	17.2 - 100	5.1	1 - 9
$P_5 \times P_2$	65.6	8.6 - 100	4.8	2 - 9

However, using the mean values of parental lines and crosses (Table 8), the correlation coefficient between the two means was 0.863. This indicates that the height of infection could be used to evaluate the disease reaction of the whole plot, but not of individual plants in the ideal disease conditions of the humidity chamber in the greenhouse. However, in the field experiments, the height of infection as a percentage of total sheath height was a more accurate measure of percent sheath blight infection and has given satisfactory results in evaluating sheath blight reaction of individual plants (Masajo, 1976). Percent infection was also used by Hashiba (1984) to estimate yield loss due to sheath blight.

The height of the highest lesions on a plant was not an accurate measure of percent sheath blight infection under the conditions of the test because a large number of apparently resistant plants developed lesions at and just below the leaf collar of some of the uppermost leaves. Therefore the lesion height was quite high, although in some cases only a single lesion was present at the collar of a leaf and the plants had no lesions on the lower portion of the plant. The appearance of the lesions on the upper leaf collar of otherwise resistant plants as discussed presently indicated that the height of infection measured as the height of the highest lesion was not a good measure of percent sheath blight infection and a good measure of disease reaction of individual plants. Therefore, it appears that under the ideal disease

conditions of the humidity chamber in the greenhouse, this lesion height is not a good measure of disease reaction.

The results obtained from this study indicate that two pairs of complementary genes affect resistance or susceptibility to sheath blight. The two parents P_1 and P_2 have the same genes responsible for susceptibility to sheath blight. The two sister lines P_6 and P_7 have similar genes controlling resistance to sheath blight. However, there is no evidence that P_5 and the sister lines P_6 and P_7 have similar genes controlling the resistance to sheath blight.

Analysis of Generation Means

The F_1 seeds planted for evaluation did not germinate. The F_2 generation means were analyzed following the North Carolina design 2 (NC2). The sums of squares of the components of variation were computed using formulas 2 through 7. The results of the computations are summarized in Table 9.

The difference in disease reaction among the male parents was not statistically significant. This indicates that all the male parents have the same level of resistance. Likewise there is no significant difference between the means of the female parents. These data confirm the results obtained from the 1979 disease nursery. The interaction between the male and female parents did show a significant difference only at the 0.1 level ($\alpha = 0.07$).

Table 9. Analysis of variance of the F_2 generation means.

Source	df	SS	MS	F
Replications	3	3.3969	1.1323	
Males	1	0.2016	0.2016	0.3347^{ns}
Females	3	1.6314	0.5438	0.9027^{ns}
Males x Females	3	1.807	0.6024	2.7124^{+}
Error	21	4.6636	0.2221	
Total	31	11.7007		

ns = not statistically significant.

\+ = significant at 0.1 level.

The formulas for the Expected Mean Squares (EMS) of the NC2 design considering all effects random are presented in Table 10. From Table 10 σ_m^2 is the variance of the male parental lines; σ_f^2 is the variance of the female parental lines; σ_{mf}^2 is the variance due to the interaction of the male and female lines; σ_e^2 is the variance of the error.

From Tables 9 and 10 the estimates of the parameters of the expected mean squares can be computed. Their estimates computed from these data are as follows:

$$\hat{\sigma}_e^2 = 0.2221$$

$$\hat{\sigma}_{mf}^2 = 0.0951$$

$$\hat{\sigma}_f^2 = -0.0073$$

$$\hat{\sigma}_m^2 = -0.0250$$

The estimates for variance due to the male parents and the female parents had negative values. This suggests that σ_f^2 and σ_m^2 are very nearly zero; and they will be considered to be zero for the rest of the computations.

Using the covariances between relatives (Becker, 1975; Hallauer and Miranda, 1980), the additive variance is estimated from formula 10 as follows:

$$\sigma_m^2 = \sigma_f^2 = Cov_{(HS)} = \frac{1}{2}\sigma_A^2 \qquad (22)$$

Table 10. Expected mean squares generated from the NC2 design.

Source	df	EMS
Replications	3	
Males	1	$\sigma_e^2 + 4\,\sigma_{mf}^2 + 16\,\sigma_m^2$
Females	3	$\sigma_e^2 + 4\,\sigma_{mf}^2 + 8\,\sigma_f^2$
Males x Females	3	$\sigma_e^2 + 4\,\sigma_{mf}^2$
Error	21	σ_e^2

assuming that there is no epistasis and that F = 1.

Since $\sigma^2_m < 0$ and $\sigma^2_f < 0$, we conclude that $\sigma^2_A = 0$. The two estimates of the additive variance have the value 0. These results suggest that the additive effects are too small to be detected. Likewise, using formula 11, the dominance variance can be estimated as follows:

$$\sigma^2_{mf} = Cov_{FS} - Cov_{(HS_M)} - Cov_{(HS_F)} = \sigma^2_D \qquad (23)$$

The solution of this equation gives the estimate of dominance variance.

$$\hat{\sigma}^2_D = \hat{\sigma}^2_{mf} = 0.0951$$

The results indicate that the genetic variance is almost entirely dominant. There is no additive variance. This suggests that a greater proportion of the character resistance to sheath blight is due to the dominance variance.

The additive and dominance variances were estimated under the assumption that there is no epistatic effect (interallelic interaction). However, it seems logical to consider epistasis present in the functioning of the genotype, whatever its magnitude is. The most important is what proportion should be attributed to epistasis. If the proportion of epistatic variance to the total genotypic variance is relatively small, the bias in the estimates caused by

assuming no epistasis will not seriously affect the selection progress.

The mean values of the F_2 progeny of the crosses $P_2 \times P_5$ and $P_5 \times P_2$ were analyzed using the paired t-test. Comparison of the calculated t statistic (1.3282) to the tabulated t value (12.706) indicated the absence of reciprocal effect in the crosses $P_5 \times P_2$ and $P_2 \times P_5$. After the first evaluation of the F_2 progeny, some of the crosses did not have enough seed to be used for a second evaluation. The data obtained from the second evaluation were not complete to fit any of the usually known designs. Therefore they could not be analyzed using the above mentioned model.

Analysis of variance was also performed by using the data available on F_2 and F_3 populations of four crosses, to estimate the genetic components of variance of individual crosses. The estimates were obtained from the following set of equations which assume no epistasis (Hallauer and Miranda, 1982):

$$\text{Variance among } F_2 \text{ individuals} = \sigma_A^2 + \sigma_D^2 + E_2$$

$$\text{Variance among } F_3 \text{ progeny means} = \sigma_A^2 + \frac{1}{4}\sigma_D^2 + E_1$$

$$\text{Variance within } F_3 \text{ progenies} = \frac{1}{2}\sigma_A^2 + \frac{1}{2}\sigma_D^2 + E_2$$

Covariance between F_2 individuals and F_3 progeny means

$$= \sigma_A^2 + \frac{1}{2}\sigma_D^2$$

Variance among parents and F_1 individuals = E_2

Experimental error = E_1

where σ_A^2 is the additive variance.

σ_D^2 is the dominance variance.

The solutions of these equations are shown in Table 11.

The data in Table 11 showed that except for the cross $P_3 \times P_7$, none of the variances are greater than E_2. This suggests that they are not statistically significant. The crosses $P_3 \times P_7$ and $P_2 \times P_5$ have negative estimates of additive variance while $P_1 \times P_6$ and $P_1 \times P_2$ have negative estimates of dominance variance.

The negative estimate of the variance may be due to the violation of the assumptions. Specifically, epistasis might be present. Alternatively, Searle (1971) suggested that inadequate sampling (small sample) and inadequate experimental techniques (competition among progenies) also might cause negative estimates of variance. Unbiased estimates of variance components are obtained from expected values of mean squares or expectations of observed variances and covariances. This implies that subtraction can produce negative estimates whenever the estimated variance is close (relative to the error variance) to zero.

Table 11. Components of genetic variance of individual crosses estimated from generation variances and covariances.

Cross	σ_A^2	σ_D^2	E_1	E_2
$P_3 \times P_7$	-2.0286	4.4206	2.25	0.4
$P_1 \times P_6$	2.3342	-4.1548	0.2122	4.2321
$P_2 \times P_5$	-0.6872	1.7464	1.42	1.71
$P_1 \times P_2$	0.1266	-0.1172	0.23	1.36

The components of genetic variance obtained from the analysis of variance of disease rating indicated a complete dominance variance for resistance to sheath blight, while the data obtained from percent infection showed completed additive effects. The discrepency between these results derived from the difference in evaluating sheath blight reaction. In fact the 0-9 scale evaluation of the disease reaction takes into account the severity and the incidence of the infection. This scale described more closely the effect of the disease on the rice plant although the human mind cannot easily differentiate rapidly between two divisions. The observations of disease reaction expressed as percent infection was not very accurate in representing the sheath blight reaction; e.g., scattered spots on the sheaths indicating 100% infection while in reality the infection is not severe. Therefore some resistant plants with few spots on the top of the sheath may be rated susceptible by the percentage method. This misrepresentation of the sheath blight reaction may reduce the dominance variance and inflate the additive variance. Therefore the 0-9 scale of rating seemed to be the most reliable in evaluating plants for resistance to sheath blight in the greenhouse.

However, in the field experiments, the percent infection has given satisfactory results in evaluating sheath blight reaction (Masajo, 1976). In estimating yield loss caused by rice sheath blight, Hashiba (1984) used the percent infection and the number of hills.

Estimation of Heritability

Breeders are usually interested in predicting the response to selection in the early generations. Therefore we will restrict our estimation to the "narrow sense" heritability estimate from the F_3 generation. Heritability was estimated from four crosses using the regression coefficients of F_3 progeny plants on F_2 parent plants (Kempthorne and Tandon, 1953; Luciano et al., 1965). The results are presented in Tables 12 and 13.

These data indicate that the heritability of resistance to sheath blight is very low. Estimates ranged from 10.83% to 16.09% when the progeny are evaluated for two weeks and from 5.69% to 12.40% when the progeny are evaluated for four weeks. The largest estimate of heritability was observed in the cross $P_1 \times P_6$ (Table 12). The F test showed that the regression coefficient was significant at 1% level in the cross $P_1 \times P_6$ and at 5% level in the crosses $P_2 \times P_5$ and $P_1 \times P_2$ when the plants are evaluated for two weeks. The regression coefficient (b) was not significant in the cross $P_3 \times P_7$.

When the F_3 progeny were evaluated for four weeks the heritability values estimated by the regression method varied from 5.69% in the $P_1 \times P_2$ cross to 12.40% in the cross $P_1 \times P_6$. However, the F test showed that none of the regression coefficients were significant (Table 13). The data in Tables 12 and 13 showed that heritability value decreased in the crosses $P_1 \times P_6$, $P_2 \times P_5$ and $P_1 \times P_2$ with two week difference in inoculation time. This

Table 12. Estimation of regression and heritability values in 4 crosses of long grain rice varieties inoculated for 2 weeks.

Crosses	n	b	$\hat{h}^2$
$P_3 \times P_7$	63	0.1624	0.1083
$P_1 \times P_6$	86	0.2413**	0.1609
$P_2 \times P_5$	92	0.1914*	0.1276
$P_1 \times P_2$	72	0.2136*	0.1424

n = number of F_2 plants per population.

b = regression coefficient of F_3 progeny on F_2 plants.

$\hat{h}^2$ = heritability in the narrow sense.

* = significant at the 0.05 level.

** = significant at the 0.01 level.

Table 13. Estimation of regression and heritability values in 4 crosses of long grain varieties inoculated for 4 weeks.

Crosses	n	b	$\hat{h}^2$
$P_3 \times P_7$	63	0.1850	0.1233
$P_1 \times P_6$	86	0.1860	0.1240
$P_2 \times P_5$	92	0.1715	0.1143
$P_1 \times P_2$	72	0.0854	0.0569

n = number of F_2 plants per population.

b = regression coefficient of F_3 progeny on F_2 parents.

$\hat{h}^2$ = heritability in the narrow sense.

implies a decrease in covariance between F_3 progeny and F_2 parents. Notice that the regression coefficients in Table 13 were calculated with the data of F_3 progeny evaluated for four weeks and the F_2 parental lines evaluated for two weeks. In such case, the relation parent-offspring is weaker than the parent-offspring relationship when both are evaluated for an equal time (four weeks or two weeks). Therefore, the heritability estimates in Table 12 are more valid than the estimates in Table 13.

In the cross $P_3 \times P_7$, the heritability value increased from 10.83% to 12.33%. But the corresponding regression coefficients were not significant. This indicates that they cannot be considered different from zero.

In estimating heritability from relationships between generations, it should be taken into account that parent-offspring regression is a biased estimate of heritability when the two generations have different means and variances. In such situations, Frey and Horner (1957) recommend parent-offspring correlation rather than regression. Heritability of resistance to *Rhizoctonia solani* was also estimated using the correlation or standard unit method. (This method establishes a heritability ceiling of 100%, in contrast to the conventional regression method which has a different ceiling for each set of data.) The heritability values calculated by the correlation method are presented in Tables 14 and 15. The data in Table 14 showed that the heritability estimates are larger than the values obtained by the regression method. They

Table 14. Estimation of correlation and heritability values in 4 crosses of long grain rice varieties inoculated for 2 weeks.

Crosses	n	r	$\hat{h}^2$
$P_3 \times P_7$	63	0.1893	0.1424
$P_1 \times P_6$	86	0.3283**	0.2462
$P_2 \times P_5$	92	0.2585*	0.1939
$P_1 \times P_2$	72	0.2406*	0.1805

n = number of F_2 plants per population.

r = correlation coefficient between F_3 progeny and F_2 plants.

$\hat{h}^2$ = heritability in the narrow sense.

* = significant at the 0.05 level.

** = significant at the 0.01 level.

Table 15. Estimation of correlation and heritability values in 4 crosses of long grain rice varieties inoculated for 4 weeks.

Crosses	n	r	$\hat{h}^2$
$P_3 \times P_7$	63	0.1233	0.0945
$P_1 \times P_6$	86	0.2351	0.1763
$P_2 \times P_5$	92	0.1373	0.1029
$P_1 \times P_2$	72	0.1343	0.1007

n = number of plants per population.

r = correlation coefficient between F_3 progeny and F_2 parents.

$\hat{h}^2$ = heritability in narrow sense.

ranged from 14.24% in cross $P_3 \times P_7$ to 24.62% in cross $P_1 \times P_6$. The correlation coefficient was significant at the 1% level in cross $P_1 \times P_6$ and at the 5% level in crosses $P_2 \times P_5$ and $P_1 \times P_2$ when F_3 progeny and the F_2 parental lines were evaluated for two weeks of inoculation (Table 14). In cross $P_3 \times P_7$ the correlation coefficient was not significant. When the F_3 progeny were inoculated for four weeks, none of the correlation coefficients were significant. This suggests that the h^2 values obtained from this estimation cannot be considered different from zero (Table 15). It should be noted also that the correlation coefficients in Table 15 were calculated from the F_3 progeny evaluated for four weeks and the F_2 parental lines evaluated for two weeks. The decrease and nonsignificance of heritability might be due to the increase in variance of F_3 progeny means. Another possible explanation might be the decrease in covariance between the F_3 progeny means and the F_2 parents.

The best effects of the two additional weeks on heritability could be shown clearly if the time factor (four weeks) were the same in evaluating the F_3 progeny and the F_3 parental lines. Since this was not the case, the heritability values in the crosses evaluated at two weeks could be considered as the best estimates of heritability in the four crosses. The heritability obtained by the correlation method (Table 14) is higher than that obtained by the regression method (Table 12). Frey and Horner (1957) explained the discrepancy by the environment which causes an expansion or contraction of the

phenotypic variability present in a population. When the environment does not cause such change in phenotypic variability the values obtained by the two methods are similar. The heritability can be increased by multiple measurements or by decreasing the environmental variance.

Implication of Inheritance Study in Breeding Methods

The results of the inheritance study indicate that resistance to sheath blight is controlled by as few as two or three complementary gene pairs. The genetic variance indicates that the additive variance is negligible leading therefore to complete dominance. According to Falconer (1981), dominance variance in such case, is maximized when $p = q = 0.5$; the genetic variance increases to a maximum at the frequency $p = 0.29$ and then decreases to 0 when $p = 1$. This suggests that as selection for resistance progresses, genetic variance will decrease without a corresponding decrease in environmental variance. As the relative importance of genotypic variance decreases, so will the expected gain from selection. In such case progeny row evaluation would be required in selecting for sheath blight resistance.

The backcross method, one of the most useful tools in the transfer of characters, has been described by Allard (1960) and Briggs and Knowles (1977) as requiring a highly heritable character. However, when the character has a low heritability, as may be the case for sheath blight, the backcross method can still be successful

when it includes a progeny test between cycles of backcrossing in order to identify plants with double dominant genes.

With the progeny row evaluation the pedigree selection can also give good results when large number of F_2 plants are selected to guarantee the inclusion of the desirable genotype among the selected individuals; the desirable genotype can then be identified more accurately in the F_3 or later generations (Sedcole, 1977; Thompson and Thoday, 1979).

Modified intermating and recurrent selection with progeny row evaluation could also be used as a breeding method. However, with the hypothesis of two to three dominant complementary genes, this method will not seem too appropriate considering the gains and effort.

SUMMARY AND CONCLUSIONS

Four long grain commercial varieties which were susceptible to sheath blight and three lines which were resistant were crossed and their F_2 progeny evaluated to determine the mode of inheritance of sheath blight resistance.

When the seedlings were inoculated and placed in the humidity chamber for two weeks the susceptible parental lines had a mean sheath blight rating varying from 6.9 to 7.8 and all had a modal class of 8. The mean rating of the resistant parents ranged from 3.1 to 3.9 and the modal class was 3 or 4.

The frequency distribution of the F_2 progeny rated for sheath blight reaction of the $P_1 \times P_2$ cross was similar to the frequency distribution of either parent indicating that these two varieties did not differ in genes for resistance or susceptibility to sheath blight. The frequency distribution for sheath blight ratings of the F_2 progeny of the crosses $P_1 \times P_6$, $P_1 \times P_7$, $P_2 \times P_6$ and $P_2 \times P_7$ was similar in each of these populations showing a bimodal distribution with modes at 5 and 7. These results indicate that the lines P_6 and P_7 have similar genes for resistance. The mean rating of the F_2 progeny of these crosses ranged from 5.5 to 5.8. The frequency distribution of the F_2 progeny of the crosses $P_3 \times P_6$, $P_3 \times P_7$, $P_4 \times P_6$ and $P_4 \times P_7$ also showed a bimodal distribution with modes at 5 and 7. The mean rating of the F_2 plants

of these crosses ranged from 5.1 to 5.6. The frequency distributions of the F_2 progeny of the reciprocal crosses $P_2 \times P_5$ and $P_5 \times P_2$ were similar and no significant difference was found between the two F_2 populations, suggesting the absence of reciprocal effects. The F_2 progeny of the two crosses ($P_2 \times P_5$ and $P_5 \times P_2$) had a mean rating of 5.1 and 4.8 and modes of 3, 5 and 7.

Infection height as a percentage of total sheath height ranged from 27 to 100% in susceptible plants and from 9 to 100% in resistant plants. A correlation coefficient of 0.439 was found between the 0-9 disease rating and the infection height. The data indicated that infection height was not an accurate method in evaluating disease reaction. The height of the highest lesion apparently was not an accurate measure of sheath blight infection since the lesions were not continuous.

On the basis of the chi-square estimation of various genetic ratios, the resistance to sheath blight appeared to be controlled by two pairs of complementary genes with resistance dominant or partially dominant over susceptibility.

The analysis of variance indicated that there was no significant difference between susceptible parents. Likewise the difference between the resistant lines was not significant. The components of genetic variance indicated that the additive variance was negligible when using the 0-9 disease rating. This suggests a high level of dominance variance and epistasis.

Heritatiblity in the narrow sense was estimated using the parent-offspring regression and correlation. The regression was performed using the rating of F_2 seedling plants and the mean disease rating of seedlings of the F_3 lines. The regression coefficients were low. The highest estimates of heritability of resistance to sheath blight was obtained in the $P_1 \times P_6$ cross. Its value was 24.13% when using the regression method and 32.83% when using the correlation method. The estimates of heritability obtained by the correlation method were higher than those obtained by the regression method. The regression and correlation coefficients were highly significant in the cross $P_1 \times P_6$ and significant in the crosses $P_2 \times P_5$ and $P_1 \times P_2$. None of the regression and correlation coefficients were significant when the F_3 progeny lines were evaluated for four weeks. However, the data could not show the effect of the two additional weeks on heritability, since the F_2 plants and the F_3 lines were evaluated for different times of inoculation.

The relatively accurate identification of the resistant and susceptible plants of the parent variety populations, with a minimum of "escapes," along with the distribution of the F_2 populations for reaction to sheath blight indicates individual resistant plants could be identified in segregating generations. However, the low heritability obtained in the F_2-F_3 line mean regression and correlation analysis suggests a need of progeny testing to obtain sheath blight resistant lines.

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APPENDICES

Appendix Table 1. Distribution of parental lines and F_2 progeny evaluated for two weeks to sheath blight disease.

Crosses	Disease Ratings									Number of Plants
	1	2	3	4	5	6	7	8	9	
P_1	-	-	-	-	3	7	9	21	12	52
P_2	-	-	-	-	3	6	16	27	21	73
P_3	-	-	-	3	7	9	16	19	6	60
P_4	-	-	-	-	9	16	19	19	13	76
P_5	5	8	24	22	2	-	-	-	-	61
P_6	-	-	20	36	19	-	-	-	-	75
P_7	2	8	27	24	11	-	-	-	-	72
$P_1 \times P_2$	-	-	-	3	18	38	55	64	39	217
$P_1 \times P_6$	-	3	18	31	43	31	35	28	13	202
$P_1 \times P_7$	-	3	11	37	47	31	34	36	10	209
$P_2 \times P_5$	-	8	40	41	52	29	31	17	6	224
$P_2 \times P_6$	-	2	20	34	43	26	26	25	13	189
$P_2 \times P_7$	1	7	44	57	54	27	30	10	5	235
$P_3 \times P_6$	1	10	24	40	47	23	27	20	7	199
$P_3 \times P_7$	-	-	3	10	21	10	10	10	6	76
$P_4 \times P_6$	-	2	15	37	63	36	43	23	10	229
$P_4 \times P_7$	1	10	31	33	52	31	32	17	5	212
$P_5 \times P_2$	-	14	46	45	63	24	24	18	4	238

Appendix Table 2. Distribution of parental lines and F_2 progeny evaluated for four weeks to sheath blight disease.

Crosses	Disease Ratings									Number of Plants
	1	2	3	4	5	6	7	8	9	
P_1	-	-	-	4	7	14	23	10	7	65
P_2	-	-	-	2	11	7	34	15	11	80
P_3	-	4	2	2	8	3	14	19	25	77
P_4	-	-	2	1	8	10	11	6	7	45
P_5	-	15	8	33	24	-	-	-	-	80
P_6	-	-	2	21	29	2	-	-	-	54
P_7	-	18	12	25	17	3	-	-	-	75
$P_1 \times P_2$	-	4	6	19	22	24	59	54	52	240
$P_1 \times P_6$	-	4	10	55	49	35	37	26	23	239
$P_2 \times P_5$	-	21	4	53	65	32	54	9	2	240
$P_2 \times P_6$	-	4	6	18	24	17	31	25	20	145
$P_3 \times P_6$	-	4	11	68	53	25	35	18	26	240
$P_3 \times P_7$	-	-	9	79	50	25	43	18	15	239
$P_4 \times P_6$	-	-	14	65	57	28	44	22	10	240
$P_4 \times P_7$	-	1	20	59	61	27	41	14	14	237
$P_5 \times P_2$	-	-	-	15	25	8	19	9	4	80
BBLE	-	-	-	-	4	9	19	17	31	80
CALORO	-	7	10	52	9	-	-	-	-	78
DAWN	-	-	-	3	11	3	26	16	21	80
ZNTH	-	16	11	25	24	3	-	-	-	79

Appendix Table 3a. Rating of F_2 plants and F_3 lines and variance of F_3 of the cross $P_3 \times P_7$ (STBN x RU7902191)

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
5	6.88	1.28
6	5.92	2.84
5	6.45	1.31
6	6.00	1.00
8	8.25	0.25
6	6.94	2.64
4	5.18	3.22
5	7.13	1.83
5	6.68	2.56
4	6.94	3.05
6	7.05	1.49
6	6.35	1.71
5	6.60	4.88
8	6.80	1.70
9	5.75	2.40
4	4.87	2.51
7	6.90	2.49
8	5.77	3.24
9	5.78	3.73
3	6.90	2.93

Appendix Table 3a, continued

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
4	5.65	1.71
5	5.55	2.36
9	6.15	2.02
9	5.80	1.28
4	7.00	1.00
5	5.65	2.87
7	7.16	1.36
8	7.80	1.74
6	7.10	3.87
4	5.47	2.37
3	7.16	2.87
3	7.35	1.17
5	6.85	2.02
6	7.52	2.26
5	6.42	1.47
7	7.53	2.43
5	4.63	4.13
6	6.80	4.31
4	5.21	2.28
5	6.72	3.21
4	5.77	0.88

Appendix Table 3a, continued

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
3	5.68	1.67
6	7.68	1.56
7	7.22	2.18
3	5.36	1.45
3	6.50	1.08
3	6.78	1.28
5	8.25	0.72
5	6.65	1.60
8	7.62	1.05
5	6.20	5.20
5	4.77	2.18
5	7.29	1.34
6	6.45	1.41
5	4.77	2.18
5	7.68	0.89
5	6.00	1.47
6	7.20	3.01
4	7.84	1.25
4	7.00	4.40
4	5.29	4.97
3	5.47	2.81

Appendix Table 3a, continued

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
7	6.73	2.31
5	7.50	1.79

Appendix Table 3b. Rating of F_2 plants and F_3 lines and variance of F_3 of the cross $P_1 \times P_6$ (LBNT x RU7902185)

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
8	8.56	0.26
7	7.00	2.00
6	5.87	2.78
3	6.11	6.10
4	6.45	0.89
8	8.00	1.50
4	7.00	2.66
5	7.83	1.79
8	6.00	2.40
7	7.73	0.78
5	6.91	1.17
3	5.83	2.16
4	7.15	1.47
6	3.50	12.50
6	8.41	0.50
8	6.91	2.08
5	6.61	1.54
8	5.82	3.40
8	6.53	3.43
6	6.75	2.50

Appendix Table 3b, continued

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
5	7.23	1.69
5	8.16	0.56
4	5.77	1.71
4	4.20	9.85
5	7.00	2.13
6	7.16	2.97
8	8.36	0.46
4	8.42	0.61
4	6.26	4.87
5	4.88	1.48
4	4.66	4.26
5	7.36	1.57
5	6.73	1.87
3	6.77	1.83
4	7.10	3.21
4	7.75	1.80
5	6.84	2.36
5	6.94	2.52
7	4.35	8.40
6	7.36	0.91
4	6.84	2.14

Appendix Table 3b, continued

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
7	5.66	0.66
7	7.55	1.79
3	6.00	1.57
4	7.00	2.33
6	6.36	1.69
3	5.15	1.81
7	8.10	0.54
6	6.84	0.91
3	6.73	2.63
5	5.64	6.24
2	6.21	1.28
3	4.25	2.25
4	6.50	4.33
4	6.00	4.73
5	6.75	2.06
4	6.05	7.27
5	7.50	2.26
4	5.00	2.28
3	6.00	1.11
5	6.25	3.14
4	2.50	0.50

Appendix Table 3b, continued

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
4	7.35	1.36
6	6.88	0.92
3	6.20	3.70
6	6.42	2.81
8	7.50	2.09
4	6.86	1.98
4	7.40	0.71
3	5.05	1.18
6	5.72	3.01
5	5.16	7.44
5	5.27	9.85
5	6.47	2.81
5	6.25	3.09
8	8.52	0.39
6	7.00	1.88
4	6.42	1.95
3	6.00	2.11
5	6.55	4.61
5	6.93	1.20
6	6.35	2.34

Appendix Table 3b, continued

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
5	5.85	2.13
3	6.00	1.64

Appendix Table 3c. Rating of F_2 plants and F_3 lines and variance of F_3 of the cross $P_2 \times P_5$ (LBLE x L201)

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
5	5.16	1.42
3	5.36	2.46
4	5.63	2.65
4	5.81	1.89
5	5.80	2.06
7	6.61	2.95
5	5.66	9.33
4	6.00	5.50
6	6.57	1.36
7	5.94	2.43
4	5.45	1.31
5	4.73	1.78
7	6.13	4.26
5	6.12	1.85
2	3.57	0.95
6	4.95	2.15
5	6.00	1.00
6	4.00	3.00
4	5.25	1.13
5	5.50	0.50

Appendix Table 3c, continued

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
4	6.66	1.09
4	5.75	2.02
3	4.50	0.50
4	6.16	3.55
3	5.94	3.93
7	6.40	3.09
5	5.00	3.33
5	3.14	2.43
5	5.41	1.88
6	5.68	1.22
3	4.21	0.33
4	6.42	4.14
6	6.72	2.33
4	5.33	1.33
6	6.00	2.00
3	4.54	1.07
6	6.64	2.99
3	4.54	3.47
3	4.52	1.59
7	4.31	0.76
4	4.50	3.14

Appendix Table 3c, continued

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
3	5.25	1.46
6	5.57	3.28
5	58.4	3.36
5	6.10	1.88
6	5.11	1.75
7	6.68	1.45
4	5.42	7.61
7	4.40	2.26
4	6.47	2.76
2	5.60	1.60
2	5.63	1.69
3	4.71	0.57
4	6.84	1.36
4	5.50	0.78
5	5.31	0.89
3	6.36	1.35
2	5.83	1.91
3	5.00	1.15
3	5.10	2.51
4	4.50	2.57
5	4.66	4.26

Appendix Table 3c, continued

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
8	7.92	5.07
3	5.44	2.02
8	6.14	1.47
7	5.93	3.92
5	3.84	2.64
8	6.76	3.19
8	5.83	2.50
8	5.12	1.58
5	5.50	2.73
5	4.52	6.38
7	5.37	2.83
7	5.00	0.90
7	6.11	1.28
3	5.57	0.61
4	5.66	4.26
4	4.20	1.70
3	5.00	7.37
7	5.13	0.98
8	5.93	3.26
4	4.83	3.08
3	6.10	1.88

Appendix Table 3c, continued

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
5	4.90	3.14
5	5.88	1.86
6	5.52	1.26
6	4.10	6.51
3	6.00	2.12
8	5.47	2.38
4	6.25	1.84
3	5.50	2.47
3	4.45	11.52

Appendix Table 3d. Rating of F_2 plants and F_3 lines and variance of F_3 of the cross $P_1 \times P_2$ (LBNT x LBLE)

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
4	8.26	1.20
5	7.62	2.91
6	5.68	1.78
7	8.00	1.05
7	7.55	1.43
6	7.31	1.22
5	7.92	0.68
7	6.94	1.34
4	6.47	2.26
8	7.87	1.31
6	7.73	1.31
4	7.18	1.36
7	7.75	1.00
7	7.57	1.25
7	7.30	3.90
6	7.42	3.14
6	6.77	4.88
8	6.95	1.73
7	7.75	1.00
7	7.21	0.95

Appendix Table 3d, continued

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
7	8.33	1.41
5	7.73	1.31
8	8.33	1.25
8	7.38	0.92
6	6.37	5.98
7	8.00	1.00
5	8.52	0.38
7	7.60	1.97
6	6.77	1.71
6	4.63	9.45
8	7.00	2.00
6	7.46	0.93
5	7.75	1.03
6	7.05	2.05
6	6.94	3.80
6	6.89	1.65
7	6.75	4.25
6	7.80	0.48
8	8.20	1.01
7	8.15	0.55
6	7.52	1.37

Appendix Table 3d, continued

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
7	6.30	5.23
6	7.15	2.64
7	7.63	1.13
5	7.71	1.45
8	7.15	1.92
5	6.61	1.08
5	7.55	1.43
8	7.80	1.64
5	8.00	0.73
6	7.88	0.61
6	7.55	2.02
7	8.21	0.61
8	8.00	1.17
6	8.15	1.36
8	7.80	0.58
6	6.75	1.65
6	8.40	0.68
5	6.35	3.47
9	7.26	2.06
8	8.53	0.60
5	7.05	3.94

Appendix Table 3d, continued

F_2 Plant Rating	F_3 Mean	Variance of F_3 Lines
6	6.50	2.26
7	8.30	0.64
7	7.25	1.58
6	5.50	5.32
7	7.63	1.35
8	6.54	2.07
8	8.13	0.55
9	7.88	0.45
8	8.35	0.74
7	6.11	1.39
7	8.00	0.55
8	6.55	2.61
8	6.94	3.55

Appendix Table 4a. Distribution of check varieties and lines inoculated for sheath blight resistance and placed in the humidity chamber for two weeks

Varieties or lines	Disease Ratings									Mean ($\bar{X}$)
	1	2	3	4	5	6	7	8	9	
Lebonnet							4	5	10	8.31
Labelle						3	5	10		7.38
Starbonnet						1	5	5	6	7.94
Leah					1	3	7	2	3	7.18
L201			4	9	4					4.00
RU7902185				4	6	2				4.83
RU7902191			1	12	4	3				4.45
RG95	7	2	4	4	2					2.57
RG94			8	7	4	1				3.90
Tetep			5	10	4					3.94
Nato			2		5	3	5	3		6.00

Appendix Table 4b. Distribution of check varieties and lines inoculated for sheath blight resistance and placed in the humidity chamber for four weeks

Varieties or lines	Disease Ratings									Mean ($\bar{X}$)
	1	2	3	4	5	6	7	8	9	
Lebonnet									20	9.00
Labelle						2	3	8	1	7.57
Starbonnet							1	3	13	8.70
Leah							1	3	14	8.72
L201		4	6	4	3					3.35
RU7902185				4	7	1				4.75
RU7902191			3	7	4	3	2			4.68
RG95	2	4	6	3	2					2.94
RG94			8	8	3					3.73
Tetep		3	4	11						3.44
Nato				1			5	2	10	8.05
Caloro		7	10	52	9					3.80
Dawn				3	11	3	26	16	21	7.30
Zenith		16	11	25	24	3				3.83

VITA

The author, Mamadou Goita, was born in 1947, in Karaba, San, Mali. He received his elementary education in M'Pessoba and finished his secondary school in Mopti. He obtained his final high school diploma from the "Lycee Askia Mohamed" in Bamako in 1968.

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EXAMINATION AND THESIS REPORT

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Major Field: Agronomy

Title of Thesis: Inheritance of Resistance to Sheath Blight in Long Grain Rice

Approved:

Major Professor and Chairman

Dean of the Graduate School

EXAMINING COMMITTEE:

Date of Examination:

December 4, 1984